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GUEST EDITORIAL

Gout

GOUT is one of the oldest diseases recorded. Its treatment by Colchicine is one of the most dramatic and specific seen in medicine. Neither the cause of Gout nor the action of Colchicine is understood.

There is much romance and many interesting stories associated with the disease. It was thought to attack only the wealthy and high livers, as a story from the Planters and Mariners Compendium will attest. This Compendium was published in 1812 and presented by Mr. Jefferson, to whom it was dedicated, to my great-grandfather:

"An English nobleman, after twenty years of riotous living, awoke one morning in the torments of the Gout. As he lay writhing with pain, his servant ran upstairs to him with great joy in his countenance: 'Oh! sir, good news! Good news! There is a famous Gout doctor below, who says he will venture his ears, he can cure your honour in a week!' 'Aye! That is good news indeed, Tom: Well, run my good boy and put up his carriage and horses, and treat the doctor like a Prince.' 'O! sir, the gentleman has no carriage and horses, I believe he walked afoot.' 'Walked afoot! What! Cure the Gout and walk afoot! Go down, Tom, go down, and instantly drive the rascal out of the house, set all the dogs upon him, do you hear? The lying varlet! Why, if he could cure the Gout, he might ride in a richer carriage than His majesty!' "

On the other hand, during the days of the W. P. A., the incidence of Gout was seen to increase in the poor men. So many men were paid for doing nothing that they had time to dissipate. The disease was sometimes called "Roosevelt's Disease".

The Virginia ham is said to owe its high flavor to the sodium urate deposits in the gouty swine. The white flecks in the muscles are allantoin, a product of uric acid metabolism.

Gout occurs over nine times as often in men as it does in women. Men with the disease are usually sexually potent until rather late in life. They even become quite proud of their disease. It has been suggested that a "Podagra Club" be founded to acquaint the gouty gentlemen of New York with each other. Strangely enough, there are a large number of doctors who have this disease.

Heredity plays a part in the development of gout. Women of gouty parents frequently run a high uric acid but do not develop the disease. We have, at present, a family of four generations, in which the great-grandson, aged twelve, has tophi and a high blood uric acid. His mother has a high blood urate but no evidence of Gout.

When Gout is seen in women, they frequently have some type of blood dyscrasia as well, such as pernicious anemia, lymphatic leukemia, or polycythemia vera. It is also seen in Lesbians.

An interesting differential point between Gout and Rheumatoid Arthritis is the reaction of the two diseases to various conditions. Acute Gout is precipitated by Jaundice,

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whereas Rheumatoid Arthritis is ameliorated by the disease. General anesthesia will precipitate Gout very quickly, while Rheumatoid Arthritis is frequently benefited. Certain emotional disturbances will benefit Rheumatoid Arthritis but will precipitate Gout. The administration of gold has been known to precipitate gouty attacks, yet gold is one of the standard treatments for Rheumatoid Arthritis. On the other hand, Cortisone and ACTH both seem to have an ameliorating effect on the two diseases. Colchicine, the drug which has the most striking effect on Gout, has no effect on Rheumatoid Arthritis.

For a number of years, drug houses have been making a preparation for the intravenous treatment of Gout. This is a 20 cc. ampoule which contains: Colchicine—1/100 grs.; Sodium Salicylate—15½ grs.; Sodium Iodide—15½ grs. The preparation has been used with success. There are those, however, who are sensitive to Iodide, Salicylates, or both. The ampoule meniscus is larger and necessitates the use of a 20 cc. syringe. Also, if split outside the vein, great pain and necrosis may result. We have seen gastro-intestinal bleeding from its use. The Sodium might be contraindicated in some cases of Gout complicated by heart and kidney disease. Recently Dr. Rice, of Eli Lilly Company, has made a 1 and a 2 cc. ampoule of Colchicine—1/100 grs. This has been given intravenously to twelve patients with acute Gout. The results have been excellent in all. The ampoule can be given undiluted, or in 500 cc. of 5% glucose. Swelling, pain, and inflammation have been seen almost to disappear within fifteen minutes of the termination of the injection. There is no diarrhea or nausea as is seen in the oral administration of Colchicine. One or two injections will usually suffice in acute Gout. If the disease is longstanding, several injections are necessary, and in some cases of chronic Gout, with acute exacerbation, Colchicine seems to have no effect. The drug cannot be used over too long a period, because it may cause liver damage. We have seen no untoward reactions with the small ampoule.

JOHN STAIGE DAVIS, JR., M.D.

Dr. Davis is chief of the Arthritis Service at St. Luke's Hospital, New York City.

Floral Eponym

Carpenteria

WILLIAM MARBURY CARPENTER (1811-1848)

Carpenteria is a single shrub of the family Saxifragaceae. It is a native of the southern part of the Sierra Nevada Mountains in California. It differs from the mock-orange only in technical characters and in being evergreen.

Dr. Carpenter was a native of St. Francisville, La. He died in New Orleans in 1948.

TREATMENT OF FRACTURES BY THE GENERAL PRACTITIONER*

O. ANDERSON ENGH, M.D.,
Arlington, Virginia.

A great percentage of fractures can be treated satisfactorily by the general practitioner. For some reason, however, he has been referring many of them to a specialist in recent years. The reason for such referral apparently is his lack of confidence in treating fractures plus a fear of litigation. The purpose of this talk is to assure the general practitioner that his fear of mismanagement, litigation or improper handling of these cases is unnecessary. Furthermore, with the thousands of physicians entering the military service there is a greater responsibility on the general physician to handle these cases. It is particularly true in rural areas.

The best way to give the general practitioner confidence is to review the treatment of fractures in various parts of the body. An attempt will be made to avoid special or spectacular methods and stress the more simple and practical ways of handling them. Some mention will be made of plates, nails, wires, pins, etc., but this will not be for the purpose of recommending the procedures *per se*, but rather to acquaint the physician with the procedures. During the course of the discussion certain dangers will be mentioned from the improper handling of cases. This is most essential since one is apt to forget certain cardinal principles and therefore jeopardize the involved part.

We will start with fractures of the clavicle. This is a fracture which the general practitioner frequently misses. It occurs often in infants and one may fail to examine the collar-bone in these patients. The fact that the child does not move the arm causes one to suspect that the trouble is in the arm or forearm and on many occasions an infant has been taken to a specialist having had X-rays of everything except the clavicle. Fortunately these fractures heal readily and, although it is recommended that as good alignment as possible be obtained, it is not so essential as in many other fractures. A "T" splint or a "figure of eight" elastic bandage or a grenadier jacket are all satisfactory ways of treating this condition. I prefer plaster of paris, even on infants, extending as low as the umbilicus and just above the elbow on the involved side. In treating this fracture, one must be sure to avoid pressure in the axilla which can occur with any bandage drawn tightly into the armpit.

Fractures of the upper humerus and about the shoulder joint: One of the most satisfactory splints is the Velpeau splint. Since this fracture occurs frequently in old people, a satisfactory result is usually obtained. The weight of the extremity even in the Valpeau splint usually produces a fair alignment. Another relatively simple method is the hanging cast which is literally a heavy cast applied from the upper arm to the hand with the elbow at a right angle. The weight of the cast usually pulls the fracture into good position. An open reduction is required only when a fair alignment has not been obtained and it is rarely necessary on aged people. Fractures of the shaft of the humerus are often treated by the hanging cast. If fair position is obtained, a shoulder spica cast can be applied.

Fractures of the elbow are dangerous. Most of these are supracondylar. These can cause the general practitioner much difficulty because he is apt to apply a tight splint or cast. A posterior mold is one of the best ways of treating this condition. An attempt should be made to correct the malposition but sometimes it is necessary to flex the elbow more than a right angle. If this is done, the splint which is applied must be loose so as to avoid pressure. An elastic bandage is preferred to ordinary cotton bandage in these cases since a cord-like pressure can usually be avoided.

Fractures of the midportion of the forearm are very difficult and may require open reduction. However, if a distracting force is applied for a considerable period some approximation will be obtained. Healing will usually take place, but the mistake to be avoided is an inadequate period of immobilization. It is often necessary to maintain the forearm in the splint for two to three months.

One of the most common fractures is the Colles' fracture. These are frequently impacted since the radius normally extends farther distally than the

*Read before the annual meeting of the Medical Society of Virginia, at Roanoke, October 8-11, 1950.

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ulna. Such a position must be regained after reduction. If the impaction has not been satisfactorily reduced, the lower ulna articulates with the carpal bones, causing a painful condition. For this reason ulnar deviation is often required. This is frequently more essential than the volar position which is stressed. One of the common complications of fractures about the wrist is causalgia. This is a reflex sympathetic dystrophy in which the patient suffers severe pain, burning, swelling and tenderness. A decalcification occurs in the bones in the forearm and the hand. It is necessary to remove the splint at once and apply one more loosely. This condition is often caused by too forceful manipulation of the fracture and often by placing the hand in a position in which there is restriction of circulation.

In fractures involving the metacarpal bones, the maintenance of the convexity of the metacarpals is essential. The use of a roller bandage in the hand with the fingers flexed over it usually gives a good result. In fractures of the fingers the tongue depressor is usually satisfactory, although fractures of the thumb require better reduction as it is so essential for good function of the hand. Bennett's fracture of the proximal portion of the first metacarpal is one which causes disability unless completely reduced. Adhesive traction to the thumb or the insertion of a small wire through the distal end of the thumb, keeping the latter in abduction, is recommended.

Fractures of the pelvis usually do not cause complications but one must always be cognizant of the possibility of genito-urinary disturbances. The use of a firm bed with sand bags to the side of the pelvis usually produces a good result and a hammock is rarely necessary. Fractures of the hip require immediate traction and intertrochanteric and subtrochanteric fractures usually are reduced by abduction and slight internal rotation and held in a hip spica cast. This procedure is not recommended as a rule since internal fixation with a Smith-Petersen nail or other form of internal fixation gives better results. Fractures of the shaft of the femur will usually heal with adhesive traction or skeletal traction using a Thomas splint. Alignment can be obtained by applying pressure where it appears indicated. Fractures about the knee joint, if not displaced, can be placed in a cast. The hip spica is preferred. If there is displacement of the fragments, it is recommended that an open reduction be performed since considerable deformity results with restricted motion, if this is not done. Fractures of the leg usually can be manipulated into satisfactory position and splints applied. Plaster of paris fixation is recommended. Fractures about the ankle are very frequent and are usually called Pott's fractures. It is very essential that the ankle mortise be maintained. These fractures are usually reduced by inverting the foot and maintaining pressure over the malleoli to tighten the mortise. Some form of internal fixation using an open reduction is occasionally necessary. Fractures of the foot usually respond satisfactorily to manipulations and the application of a cast.

Inasmuch as it is necessary to apply plaster of paris frequently in fractures of the lower extremities sufficient padding, using felt, is required. One of the most common complications is an ulcer over the heel. If the patient complains of even a slight amount of burning this portion of the cast should be removed since it can produce a pressure sore. This may demand skin grafting. One of the other complications of traction and splints to the lower extremities is in the region of the fibular head. Pressure on this area will produce a peroneal paralysis which is transient ordinarily but in some cases may be permanent.

In fractures of the spine, hyperextension is usually indicated. At the time the patient is first seen he should be maintained in this position unless it produces some evidence of cord or nerve pressure. A firm bed, a firm mattress or the insertion of boards between the springs and mattress is essential. One or two pillows should be inserted underneath the area of fracture. In the cervical spine a small roll can be inserted. Uncomplicated fractures of the thoracic and lumbar spine can usually be treated by the application of a cast or brace within a period of a few weeks, although there is a tendency recently to permit these patients to walk without some form of support. It is recommended that they wear a body cast for a period of approximately three months and a corset for an additional three to six months.

This is a simple review of methods which are used at this time and in many respects do not differ from what was taught twenty-five years ago. It is most essential that one recognize the principle involved and if this is done one is not apt to make any serious errors.

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In conclusion, it is felt that the general practitioner can satisfactorily treat a great percentage of fractures if he follows fundamental principles. Most fractures can be treated by simple measures, avoiding forceful manipulation and requiring adequate but not restricting immobilization.

DISCUSSION

DR. CHARLES J. FRANKEL, Charlottesville: I think Dr. Engh is to be congratulated for several things. The first is his humility in coming down off the pedestal which the usual orthopedist or the usual specialist occupies.

I agree with Dr. Engh in most respects, but I disagree in several. First, I think he has oversimplified the problem. The handling of fractures is not quite as simple as Dr. Engh has told you. There are many complications to fear, and I am afraid that he has a great deal more faith in human nature and in some people than I have. I think if you will examine the legal records you will find that as the economic cycle changes people have more and more tendency to sue. If times are good people do not sue. If we are to continue to have the "Fair Deal" in Washington, I think we can expect an increase in suits. So we must be careful about leaving ourselves vulnerable to medicolegal action.

It is perfectly true that about ninety per cent of the fractures can be handled by conservative methods. About ten per cent require operative procedures. Of the ninety per cent, I should be inclined to tell the general practitioner to sit down and classify the type of fractures that he thinks he is capable of handling. I do not think he ought to try to cover the whole field; there are many fractures that he ought not even to bother about. Among those fractures are fractures about the hip. It is perfectly true that the general practitioner can handle intertrochanteric fractures, but the mortality among these aged people is terrific. The fracture may heal, but the patient may not live long enough for it to heal. The incidence of pneumonia and other complications is fairly high among elderly patients who are kept in bed and have not the advantages of good nursing care.

I think one of the most common mistakes made not only by general practitioners but by specialists is the failure to take X-rays in proper sequence. There must be, for diagnostic reasons and medicolegal reasons, an X-ray before the fracture is set, an X-ray after the fracture is set, and then a follow-up X-ray a week or ten days after the fracture has been set. That does not mean you have to take pictures of the nondisplaced fractures, but for the common type of displaced fracture you had better check a postoperative film in about a week or ten days. Make that a routine. If you are going to handle fractures, handle them by routine, so you can know what to do.

A second mistake, I think, is failure to interpret X-rays. Those of you who handle those fractures sometimes are called upon to interpret your own X-rays, and with lack of experience you may misinterpret them. I should like to say to you, if you are in an area where you cannot get expert X-ray interpretation, don't hesitate to take an X-ray of the normal side. You may have to take an extra film, but then you have the X-ray of the normal side for comparison.

The next mistake is under-immobilization or over-immobilization. I have seen simple fractures of the elbow in children immobilized for as long as thirteen weeks, resulting in ankylosis. It is true we get ankylosis sometimes without too long immobilization, but the chances for it with over-immobilization are greater.

Another mistake on the part of general practitioners is over-optimism. There is a tendency to tell parents that everything is going to be fine. On occasion that is justified; on other occasions it is not justified. Where you have involvement of epiphyses or joints, I do not think you are justified in telling patients that everything will be all right. A patient with involvement of the epiphysis or involvement of the joint may not have any bad results at all, but he may develop arthritis at a later date.

In setting fractures it is necessary to have assistants. That is an advantage we have in the hospital. It is almost impossible to do a good job if you are going to do it alone and simply have a nurse give the anesthetic for you. If you can get any help, get it; you can tell the other person what to do. But don't try to do it alone.

I cannot quite agree with Dr. Engh when he suggests that you handle supracondylar fractures. If a supracondylar fracture is displaced, I think you had better get rid of it. There is an old German proverb which says: "It is better to be a barking dog than a dead lion."

Plaster of paris is not a very difficult thing to apply, but there are certain fundamentals which you must master. You must know how to put it on; you must know that it has to go above or below the joint; you must know that certain points have to be padded; you must know that certain joints have to be immobilized. Those things are available to us in textbooks, but we get so busy we do not look them up. Some of us hesitate to look up things in books, but I think that is wrong. I certainly look into an anatomy textbook before I go into the operating room.

When the general practitioner turns a case over to the specialist, I think he ought to keep an interest in that case. I do not think the specialist ought to take over a case and forget about the general practitioner's interest. I think that the follow-up and the end result of the case ought to be gone into with the general practitioner, so that both he and the specialist maintain a mutual interest in the patient.

One other thing Dr. Engh mentioned is the handling of fractures of the scaphoid, in the wrist. I should like to impress upon you the necessity of taking the X-rays in four views. You cannot in most cases diagnose simple fracture of the scaphoid by taking anteroposterior and lateral views. You must take four views; and you will find, in taking those four views, that about thirty per cent of the fractures will become visible, whereas they are invisible in the average anteroposterior view. There again you must rely on your interpretation of the X-rays.

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If you are going to handle difficult fractures, then you must be ready to accept the responsibility. I think that if you handle these with the conservatism, which is the rule, you will get better results than if you try to follow the tendency toward the use of hardware. Most of the men who finished in Army hospitals came out with almost a savage lust for the use of hardware and metal. That may be all right in the Army hospital, where the patients have close supervision; but it is not so good in the average civilian hospital. We believe in our hospital that conservative measures can be used in over ninety per cent of fractures.

I think Dr. Engh is to be congratulated upon a very clear and concise statement of the fundamentals in the treatment of fractures.

DR. MARCELLUS A. JOHNSON, JR., Roanoke: I just want to bring out a couple of points in the discussion. I enjoyed the doctor's paper. Maybe I am not qualified to talk about it. In our clinic, we have over eighteen hundred fractures a year. Many things stand out very prominently.

About the general practitioner, I deal with a lot of them, and I should like to say this. He has got to use the same judgment as he uses about everything else. He becomes only an A & D officer, as we said in the Army—an admitting officer—if every time he has a pain in an abdomen he refers it to the surgeon and every time he has a fracture he refers it to the orthopedic surgeon. I think the general practitioner is to be trusted to use judgment in a bone case just as he is in an abdominal case, and that he knows when he ought to get help. Otherwise, he becomes only an A & D doctor.

We are considered by the courts as medicolegal experts. Having passed the State Bar examination of Virginia, there are a number of things I have picked up in court. You would be surprised to find how many times doctors are sued or threatened with suits. Most of the cases against doctors are because of ill-timed remarks. I would call to your attention that we all have to be considerate of the other man. Last week I had two fracture cases that an orthopedic man had set, Colles' fractures, comminuted, very severe. They were not in good position. We, at our clinic, check practically all fractures every two weeks. Key and Conwell and other good books will tell you that two out of twenty will slip. These two fractures were set by orthopedic men, and the X-rays are on record and can be shown. They were not in good position. I had a good out, and that is that some do slip, even if correctly set. But I think these two slipped because the orthopedist left it to his assistant to put on the cast and the cast was not put on above the elbow.

Every man who belongs to the medical profession ought to be thoughtful and very careful about what he says, and should not make thoughtless remarks. A doctor has to use good judgment in a bone case, just as he has to in an obstetrical case or in abdominal pain or head pain, and he should be very courteous, and very thoughtful of his brother doctor.

DR. MARTIN B. HIDEN, Warrenton: I am a small-town surgeon, and I have to handle some fractures. I believe that the general practitioner and the general surgeon and all the rest except the orthopedic surgeon would be better off if they did not handle fractures, because they have more dynamite in them and are more dangerous than any other class of practice I know. If all of us who are not orthopedic surgeons could get by without handling any fractures whatever, we would benefit. When it comes to handling fractures I am in the same class with the general practitioner; I do not know any more about it than the general practitioner, although he thinks I do. If the orthopedist could handle all of them we should all be happier because some of the simplest fractures, or what look to be some of the simplest fractures, can cause an awful lot of trouble.

When I suggested this title, I wanted the orthopedist not to tell us what to treat and what not to treat, but what to do and what not to do until we can get help.

One thing I might mention is head injuries. It seems to me that one of the things the family of the patient always emphasizes is, "Is the skull fractured?" If you can tell them that the skull is not fractured, then they are apt to think that the patient is not injured very much. But the damage to the brain is what counts, not the damage to the skull. There can be fracture of the skull without symptoms. Tell the insurance people and others about the damage to the brain, and minimize the fracture of the skull. Talk in terms of damage to the brain—laceration, bruises, or whatever you have. Don't let them get the idea that because the skull is not fractured it may be very much simpler, or that if it is fractured it may be more grave. It may be that you cannot see fractures at the base of the brain, and it does not make a great deal of difference whether you do or not.

The same thing applies to fractures of the spine. Emphasize the damage to the spinal cord rather than the fracture or dislocation of a cervical vertebra. Sometimes that is very hard to diagnose. It may be that you cannot see the fracture. It does not always make a great deal of difference. What you are up against is damage to the cord.

As to fractures in general, I cannot see where extension would do any harm in the majority of cases. It often does a great deal of good, and it is a great thing for the patient from a mental standpoint, because you are doing something. In a great many cases of dislocation, if you can get rid of the spasm in the muscles by extension, the dislocation will probably go back under its own power.

I do not always know how to put my splints on, or how to put my casts on correctly. Sometimes I get into more trouble with the circulation than I do with the fracture. Either I leave the cast on too long, or I have it a little too tight, or I do not start passive motion soon enough. I do not get contractures, but I get a stiff hand in Colles' fracture and it has that glazed look; and it takes six months to get well instead of six weeks.

The way we handle fractures in my territory is that if a general practitioner gets a fracture which he thinks is simple enough, he treats it himself—and usually treats it very

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well. If he had a list of "Do's" and "Don'ts", it would help him. If he suspects that he should not treat it, then he calls me, and I usually call an orthopedic surgeon. I wish I could call him on all of them.

When it comes to a suit or a bad result, people ask, why did you not call in an orthopedic man, or, why did you not call a specialist. The implication is that if you had called a specialist you would probably have gotten a good result. Maybe you would, or maybe you would not. Why didn't you call him if you thought a bad result was possible or probable? The reason you did not call him is because you wanted to hold on to that patient or wanted to save the patient expense. Neither of those is a very good reason.

If general men want to avoid trouble, they should treat as few fractures as possible, and, when they refer them to me, I think I should treat as few of them as possible. I had better refer them to an orthopedic surgeon. The orthopedist should take the general practitioner into his confidence and tell him what to do and what not to do. Tell him what to do until he can get an orthopedic man there. On Saturday afternoon or Sunday you have a difficult time getting an orthopedist, because he is out playing golf or doing something else. If you have a charity case, I think I can treat it better than their interns.

DR. ENGH, closing the discussion: So far as the general practitioner is concerned, I think his judgment is good enough that if he thinks he is going to run into trouble he will usually call for help. This talk today was given with the hope of stimulating interest in this subject among general practitioners. That is the only way that the general practitioner can continue to make progress, and also to review the principles from time to time.

Sponge Biopsy.

Sponge biopsy, a newer method of cancer detection, has proved valuable in discovering cancers of the cervix, rectum, mouth and skin. It is described by Dr. Sidney Gladstone of New York in the April 21st *Journal of the American Medical Association.* Dr. Gladstone is associated with the New York Polyclinic Medical School and Hospital.

The procedure is fairly simple. A gelatin or cellulose sponge is rubbed on a suspected lesion. By rubbing, small particles of tissue are picked up by the sponge. These particles are then examined by a pathologist for possible cancer cells.

"For the detection of early cervical carcinoma [cancer of the lower part of the womb], the method of sponge biopsy was found to be very reliable and distinctly superior to the method of surgical biopsy as usually performed. [Surgical biopsy involves removal of a small piece of suspected tissue for examination.] Because of the absence of distinctive visible changes, it is not surprising to learn that a single small block of tissue removed by surgical biopsy from the most favorable site will miss half these lesions."

Dr. Gladstone has done over 1,200 sponge biopsies in two years. In a series of 641 women examined for cancer of the cervix uteri, he found 16 cancers. Nine of these were suspected because of ulceration, bleeding or both. Seven cancers were early, however, showing no symptoms. Sponge biopsies on 32 patients with ulcerating lesions of the rectum showed cancers in 15.

"These findings were all confirmed by surgical biopsy, surgical specimen or operative findings. In two cases with correct diagnosis of cancer by sponge biopsy, the surgical biopsy specimen had failed to include cancerous tissue and was falsely interpreted as negative. In our experience, sponge biopsy has succeeded in collecting diagnosable cancerous tissue in all cases of cancer of the cervix and rectum."

Dr. Gladstone further reports 40 sponge biopsies on ulcerating lesions of the mouth, revealing 12 cancers, and 22 sponge biopsies of the skin, indicating 11 cancers—all confirmed by surgical biopsy.

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INTESTINAL OBSTRUCTION*

CHARLES W. PUTNEY, M.D.,
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Intestinal obstruction is of paramount importance to the physician and surgeon alike. One who is confronted with a patient with acute abdominal pain and acute constipation, nausea and vomiting and with high-pitched tinkling sounds and audible peristalsis, must alert himself to the many conditions that produce one or more of these symptoms. However, when these symptoms are noted, obstruction must receive first consideration and prompt treatment must be instituted. In less acute cases, more time can be taken for a more complete study. In the proper approach to these cases, sign posts may be noted which could lead to the diagnosis of many conditions. Following a careful history and physical examination, other studies should be made, including blood and urine examinations. A scout film or flat plate of the abdomen may reveal a dilated, ballooned colon so characteristic of a volvulus and the diagnosis can be confirmed by a barium enema; or it may show fluid levels or step ladder dilatations, presenting the characteristic bowel pattern of small bowel obstruction; or viscera may be found in the chest, indicating a diaphragmatic hernia. If these are not observed, suggesting an obstruction, calciferous deposits may be seen in a dermoid cyst of the ovary or fibroid uterus, or shadows may be seen suggesting a fecalith in the appendix or stones in the biliary or genito-urinary tract which may lead to the correct diagnosis. Gas shadows under the diaphragm in a patient with abdominal pain, associated with shoulder pain, will confirm the diagnosis of a perforated peptic ulcer. Blood and urine studies may reveal findings suggestive of certain constitutional diseases of interest to the internist and may assist the surgeon in evaluating the risk. An electrocardiogram on a patient around forty years of age or older may give evidence of coronary disease.

It is impossible to cover the entire subject of intestinal obstruction, so I shall only attempt to present certain points that have proven of value to me and trust that by so doing it will serve to renew your interest in this subject. In presenting this paper, it is not my intention to throw out a barrage of scientific terms and build up an oral fortification behind which to hide my ignorance, nor to speak as an authority; rather it is as one who has struggled with these patients for more than 25 years and have watched the mortality rate fall from more than 50 per cent to less than 10 per cent by the use of improved methods. During the first five years of this period, we still had to use impure rubber tubing for intravenous therapy, with untoward reactions. During the next ten years chemically pure tubing was available and we had fewer reactions. For the past ten years the pharmaceutical houses have supplied us with disposable containers and reactions have been rare, unless we become over anxious and literally drown a patient with saline or allow it to run in too fast on a hypertensive patient. This is less apt to occur, however, in treating obstruction, due to the rapid loss of chlorides and fluids. During this last period, the Levin and the Miller-Abbott tubes have come into more general use and have been made more efficient by the Wangensteen negative pressure apparatus. We must not forget that neither vomiting nor the Miller-Abbott tube can empty the contents of a closed loop of obstructed bowel.

Biochemists have given us a workable knowledge of the electrolytes and have shown us the value of cross matching blood, Rh factors and of the electrolyte fluid balances. Our technicians have become so efficient in this work that we can usually keep these elements around a normal base level by intravenous therapy. This teamwork, together with the sulfonamides and antibiotics have contributed to better surgery and reduced mortality.

The responsibility lies with the practitioner who first sees the patient with symptoms of an acute intestinal obstruction. It is he who may sign the death warrant, by giving narcotics and cathartics, whether he signs the death certificate or not. Procrastination is more dangerous in these cases than exploration, and may mean death. These patients should be immediately referred to a competent surgeon. Any lapse of time should be utilized by x-ray and laboratory

*Read before the annual meeting of the Medical Society of Virginia at Roanoke, October 8-11, 1950.

studies and securing sufficient compatible blood, or having suitable donors standing by, and giving intravenous glucose in saline and starting negative pressure.

Neurotic females should be viewed with a critical eye. One patient in my experience, a normal, though apprehensive girl with all the classical symptoms of an acute obstruction, had a copious evacuation in bed just as the stretcher came to take her to surgery.

A similar patient with a history of four previous abdominal operations was being observed. She wished to change surgeons to "get something done", was operated upon by one of my colleagues but no obstruction was found. These neurotic patients rarely have obstruction and are not benefited by surgery, which usually adds to the confusion of the next surgeon.

TYPES OF INTESTINAL OBSTRUCTION

1. Mechanical obstruction
2. Paralytic obstruction
   - (1) Acute
   - (2) Chronic
   - (3) Chonic, becoming acute

CAUSES OF ACUTE INTESTINAL OBSTRUCTION

(A) Obstruction with strangulation (62%)

1. External hernias (45%)
2. Internal hernias (2.5%)
3. Intussusception (15%)
4. Volvulus (2.6%)
5. Bands (11.4%)
   - (a) Peritoneal adhesions
   - (b) Meckel's diverticulum
   - (c) Appendix
   - (d) Great omentum

(B) Obstruction without strangulation (38%)

6. Adhesions (7.4%)
7. Congenital malformations (.6%)
   - (a) Atresia of small gut
   - (b) Atresia of large gut
   - (c) Imperforate anus
8. Impaction of gall stones (.7%)
9. Impaction of foreign bodies
10. Impaction of round worms
11. Growths of intestines
12. Diverticulitis
13. Fibrous strictures
14. Pressure from without
    - (a) Tumor
    - (b) Endometriosis

CAUSES OF CHRONIC INTESTINAL OBSTRUCTION

A. Extrinsic
   1. Adhesions
   2. Pressure from without
      - (a) Tumor
      - (b) Endometriosis

B. Intrinsic
   3. (a) Inflammatory
      - (b) Traumatic
      - (c) Neoplastic
   4. Carcinoma of colon
   5. Carcinoma of rectum
   6. Chronic diverticulosis
   7. Chronic intussusception
   8. Chronic volvulus
   9. Ileocecal tuberculosis
   10. Ileocecal actinomycosis
   11. Fecal impaction
   12. Hirschprung's disease
   13. Chronic regional ileitis

CAUSES OF PARALYTIC OBSTRUCTION

(A) Paralytic Ileus
   1. Inflammatory: as peritonitis
   2. Toxic: as uremia, lead poisoning
   3. Traumatic, postoperative
   4. Neuropathic:
      - (a) Lesions or pressure on spinal cord.
      - (b) Lesions or pressure on peripheral nerves.

(B) Mesenteric vascular occlusion
   - (1) Embolism or thrombosis-superior mesenteric vessels
   - (2) Portal cirrhosis and infection.

SYMPTOMS OF ACUTE INTESTINAL OBSTRUCTION

1. Pain is severe, with abrupt onset, becoming colicky and continuous. The pain may be in the back in cases of volvulus of the small bowel.
2. Nausea and vomiting, repeated at first; later, it is less frequent and fecal.
3. Constipation is complete after the first half hour. In subacute or chronic cases, the symptoms are less marked.

PHYSICAL SIGNS OF INTESTINAL OBSTRUCTION

1. Shock is not marked unless strangulation is present.
2. Distention is present over the central portion of the abdomen and tympanites is present.
3. Gas and feces return with the first enema but none after an enema, if properly given.
4. Tenderness and rigidity are absent at first but present after peritonitis develops.
5. High-pitched tinkling sounds and audible peristalsis are present early but may disappear after gangrene develops.
6. Other abdominal signs may be absent though tumors or masses may be felt. External hernia may be found. Scars may or may not be present.
7. Late signs: Peritonitis brings leukocytosis and elevation of temperature. Dehydration brings pallor, sunken eyes, dry tongue and loss of chlorides, etc.; most marked in high obstruction, while distention is more marked in low obstruction.

Intestinal dilatation alone seems to have a deleterious effect and may lead to ultimate death (shown by experimental tests on dogs), without loss of fluids or electrolytes, as they accumulate and are retained inside the distended bowel and outside of the vascular system. This toxic material can usually be removed by the Miller-Abbott tube and the Wangensteen nega-

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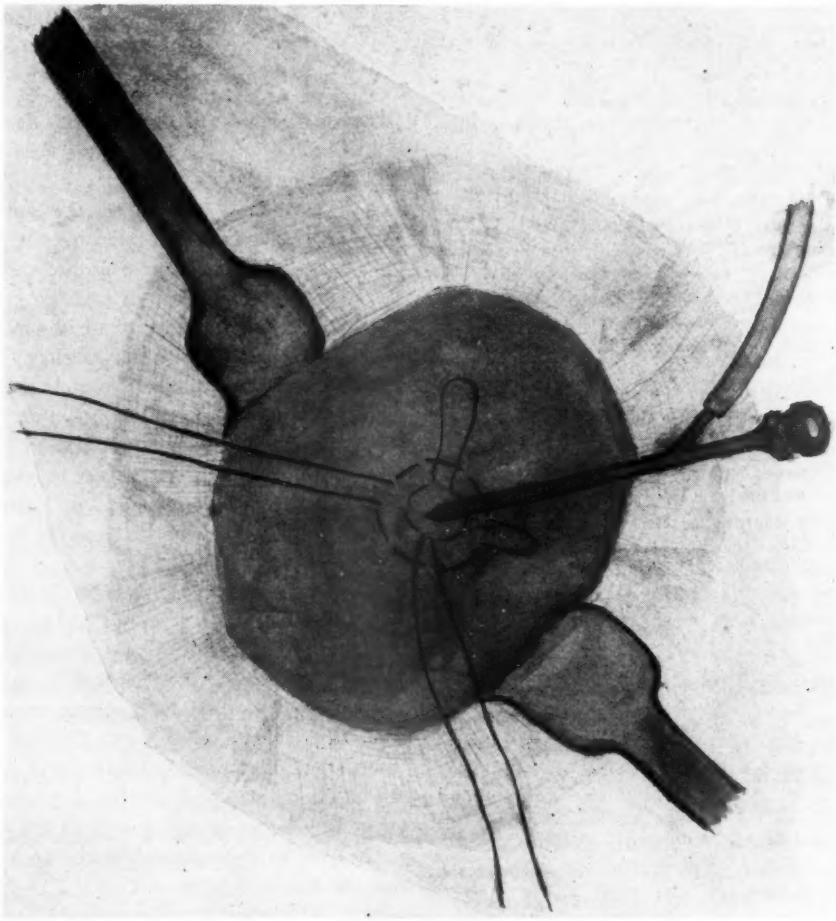
tive pressure apparatus, except in a closed loop. Fluids and chlorides must be replaced into the circulation or certain physiological changes will result:

1. Hemoconcentration with shrinking of plasma volume.
2. Reduction in the fixed base of the serum.
3. Diminished plasma chlorides.
4. Increase in blood urea and non-protein nitrogen.
5. Increased carbon dioxide combining powers of the plasma.
6. Urine is often acid and may contain ketone bodies, and may show marked reduction in chloride content.
7. Later alkalosis develops if vomiting persists and if the electrolytic fluid balance is not restored.

Tests for these changes should be made and, if alkalosis develops, two per cent ammonium chloride solution may be given intravenously to combat convulsions and other symptoms of alkalosis.

Diagnosis is based on a history of an acute onset of severe abdominal pain, followed by nausea and vomiting, constipation, high-pitched tinkling sounds and audible peristalsis, not completely relieved by an enema. Distention is present over the upper central portion of the abdomen but may be general. The diagnosis may be confirmed by the findings of a strangulated external hernia or by x-ray which will reveal distended bowel, with characteristic bowel pat-



This drawing is to illustrate how a distended gut can be deflated at operation by the use of a trocar or suction tip attached to a suction machine.

tern above the obstruction but no gas shadows below, after an enema.

It is not within the scope of this paper to give a complete differential diagnosis of intestinal obstruction but only to stimulate our thoughts on certain possibilities, as more mistakes are made by not thinking than by not knowing.

External irreducible hernias are usually readily found by a good history and careful physical examination. Femoral and obturator hernias may be overlooked by a hasty observer.

Internal causes are more difficult to diagnose. Intussusception should be suspected especially in children and other acute cases when blood and mucous are found in the stools.

An infant that vomits bile in the first few days of life and has a distended, shiny abdomen, with dilated abdominal veins, tympanites and obliterated liver dullness, without milk curds in the stools, should be x-rayed for evidence of duodenal or intestinal atresia. Congenital anomalies of the lower gastro-intestinal tract are fairly uncommon but when found should be operated during the first 48 hours of life. The lesions may be multiple, and atresia, stenosis or malrotation may be found. X-ray will assist in the diagnosis of these distended infants. An imperforate anus may be found on inspection.

Functional intestinal obstruction on a congenital neurogenic basis in infancy has been described by Zueler and Wilson. There is a familial tendency and may be due to failure of development of certain nerve tissues or absence of nerve cells of the plexus of Auerbach (myenteric plexus); or have, as has been shown in some cases, so-called idiopathic megacolon. Symptoms begin at birth or in the next few weeks and are those of intestinal obstruction, but, at operation, no obstructing lesions are found. Symptoms are similar to those of paralytic ileus but have active and audible or visual peristalsis. The anal sphincter may be unusually tight. The feces, if passed, are hard-like pellets and may alternate with diarrhea. X-ray pictures are similar to those of mechanical obstruction. Treatment is exploratory laparotomy, with low enterostomy and an attempt at closure later. Dilatation of the anus should be done while the patient is under anesthesia.

In adults, obstructing scars or lesions of the pylorus, duodenum or jejunum may produce symptoms of high obstruction, rapid dehydration and loss of chlorides with nitrogen retention. Alkalosis may develop.

Gall stones that have caused perforation of the gall bladder into an adherent gut may cause an obstruction. Dr. C. W. Mayo has reported 18 such cases. However, they do not all obstruct. One of my patients passed two large gall stones without obstruction.

Early post-operative obstruction is usually due to inflammatory adhesions. Late post-operative obstruction of the small intestine is usually due to adhesive bands. Vomiting occurs early, repeated and copious. Adhesive bands may or may not be due to previous surgery. The absence of an abdominal scar should not lead one astray in the presence of symptoms of obstruction; and in the presence of a scar one must not assume that adhesions are necessarily the cause. The author has seen one patient with adhesions and obstruction from a bullet wound received 39 years before. X-ray revealed the bullet in the second lumbar vertebrae and only a small abdominal scar was seen.

Volvulus of the sigmoid begins with a sudden colicky pain. X-ray reveals a characteristic ballooned dilated large bowel above the obstruction and the bowel rarely holds more than a 500 c.c. enema. The barium enema comes to an abrupt stop as shown by x-ray. Acute mechanical obstruction of this type begins with an abrupt onset of continuous colicky pain, with vomiting, shock and evidence of a catastrophe. Delay is hazardous in these cases.

In subacute or chronic large bowel obstruction, about 90 per cent are due to carcinoma. They usually have a history of change of bowel habits, are past middle life and usually have blood in the stools. Carcinoma of the large bowel often gives a history and symptoms of progressive constipation, alternating with diarrhea. Blood changes are not marked in carcinoma of the large bowel until late. Anemia is often marked in carcinoma of the right colon by the time the diagnosis is made as it usually causes less obstructive symptoms in this region. Large bowel obstruction due to tumor produces intermittent cramp-like pain; and may have episodes of loose stools with the first few attacks. No elevation of temperature or pulse rate is noted. Vomiting is late and may not occur if the ileocecal valve is competent.

Endometriosis may compress the lumen of the gut and cause obstruction. Often bloody diarrhea

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with periods of attacks of abdominal distress is noted between the periods of attack and menstrual periods with dysmenorrhea. Endometriosis is the only common condition in which there occurs invasion of one tissue by another normal tissue of the same host. Usually there are signs of extensive endometriosis and signs and symptoms of intestinal obstruction.

Digital examination is of value to differentiate between paralytic and mechanical obstruction. If mechanical obstruction is present, the bowel is collapsed against the examining finger, while paralytic bowel presents a dilated ampulla of the rectum following irrigation.

Paralytic ileus causes distention of both large and small bowel, shown by x-ray and audible peristalsis is not present. It is usually due to post-operative peritonitis or traumatic shock but may be due to debilitated conditions such as uremia, lead poisoning, etc. Paralytic obstruction may also be due to peritonitis from a ruptured appendix, perforated peptic ulcer or from intestinal biliary colic, renal and urethral stones, tortion of ovarian cyst, pulmonary infection, tabes dorsalis, tumors or lesion of the cord, etc.

Treatment: (1) Relieve the distention and evacuate the bowel. (2) Treat or remove the cause but a shunting operation may be necessary. (3) Resect all gangrenous or non-viable small bowel and do an anastomosis. (4) Exteriorize or resect gangrenous large bowel.

It matters not how urgent the indications may seem, one should start the Wangensteen suction; and make an effort to restore some of the electrolytic fluid balance by starting intravenous saline, followed by blood transfusion, continuing on through and following operation, until fluid balance is restored.

If external strangulation or incarcerated hernia is found, delay is inadvisable and operation should be done immediately. Otherwise, scout films are taken on admission if acute obstruction is suspected. Other films are taken as indicated. Laboratory studies should be ordered, consisting of urinalysis, including chloride estimation, acetone and diacetic acid; complete blood count, Kahn, blood chlorides, blood urea, serum protein, blood typing, cross matching and at least three pints of blood should be immediately available. The patient is given two ounces of mineral oil by mouth or through a nasal tube and a two quart enema is given. If the colon will not hold this amount of fluid, a barium enema should be given in search of low obstruction, volvulus, etc. The amount of distention is measured and noted. Intravenous 5 per cent glucose in saline is started. A nasal tube is inserted and negative pressure is started. The tube is left open two hours and clamped off one hour, and repeated continuously. Clear liquids and an ounce of mineral oil is given each time the tube is clamped off.

A saline enema is given every two hours and search is made for oil on the return fluid. If mineral oil does not return with the saline enema after a few hours, and the distention is increasing and the patient's condition seems less favorable, immediate operation is indicated. The higher in the gastro-intestinal tract the lesion is, the greater is the indication for immediate surgery, due to more rapid dehydration. The lower in the gastro-intestinal tract the lesion is, unless strangulation is present, the longer we may wait to get a good work-up of the case and more time can be taken to get the patient deflated and ready for surgery. One gram of sulfasuxadine or one-half gram of sulfathaladine should be given by mouth each time the nasal tube is clamped off, every three hours for five days if the patients condition seems satisfactory. This presumably is partly wasted and cannot enter a closed loop, but practically it seems to favorably influence post-operative results. The author has also used streptomycin (1 gram by mouth, b.i.d.) the same way for two days before surgery with satisfactory results.

Supportive treatment is given to overcome dehydration and restore electrolytic balances by intravenous saline, 5 per cent glucose, plasma, blood transfusion, protein solutions, etc., and to restore chlorides and serum protein. Enough fluid should be given to recover at least 1,000 c.c. of urine each 24 hours. Distention must be overcome. The Wangensteen negative pressure apparatus, using the Levine or Miller-Abbott tube is usually very helpful pre-operatively and may be adequate in paralytic ileus. The Miller-Abbott tube, however, cannot empty a closed loop and too much time should not be lost but used only to the point of maximum results and only until the patient is properly prepared for surgery. If the temperature and pulse become increased, the abdomen becomes tender, or peristalsis ceases, the patient is immediately decompressed by surgical means. The type of operation depends on

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the condition of the patient and the cause and condition found when the abdomen is opened. If dark, bloody fluid is found in the peritoneal cavity, we must look for gangrenous bowel and, if found, it must be removed. If resection of the non-viable small bowel is not done, they all die, according to McNeiley, Chief Surgeon at the Cook County Post-Graduate Medical School, and this has also been my experience. The operation must not be postponed in very acute cases. In 98 per cent of the cases, the causes of obstruction can be corrected at operation without resorting to enterostomy. If the bowel is not gangrenous, troublesome distention may be relieved at operation by placing a pursestring in the bowel and inserting a trochar or suction tip into the distended bowel. Strangulated gut demands immediate surgery whether due to volvulus, hernia, or whatever cause. Gangrenous large bowel can be exteriorized and removed according to Mickulitz technique. The obstructing lesion should be removed at the time of operation if possible. It may be necessary to do a shunting operation or an enterostomy or colostomy above the lesion, unless gangrene is present. All non-viable bowel must be removed from the abdominal cavity. If resection is done or if there is possible contamination, it has been my practice to place 1 gram of streptomycin in solution into the abdomen just before closure.

In cases of repeated recurrences, intestinal plication may be successful. In cases due to malignancy or tumors of the large bowel, they should be more carefully prepared by giving sulfasuxadine or sulfathalidine, orally for 4 or 5 days before surgery and streptomycin 2 days before the resection is attempted. It is given in other cases if time permits. A preliminary transverse colostomy is done for lesions of the left colon or cecostomy for lesion of the right colon, followed by resection several days later after the patient is properly prepared.

If endometriosis is the cause of obstruction, both ovaries should be removed to prevent menstrual stimulation of this tissue. The obstructed bowel may have to be resected and a panhysterectomy may have to be done. X-ray treatment may be of value in incurable cases of endometriosis.

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DISCUSSION

DR. CARRINGTON WILLIAMS, Richmond: This brings up such a variety of approaches I will only try to cover a few of the points Dr. Putney has made.

In external hernia, especially in elderly women and especially those who are fat, it is not at all difficult to miss a small femoral hernia. I have seen several that puzzled me for awhile until I was careful enough to palpate the groin. Any patient with acute abdomen should be carefully examined for hernia. Even at times if you feel a lump you may be confused whether it is a small strangulated hernia or some other type of mass. The obstructions from adhesions is awfully interesting. It is remarkable to see how adhesions can stay so long before causing trouble. I had an old lady recently who had been operated on 42 years before; the adhesions had given her a complete obstruction after this long time.

I would like very briefly to discuss the Miller-Abbott tube. It seems to me in some clinics today they are paying a little too much attention and effort to preparing the patient and delaying the operative procedure. Now, all of us agree that they should be restored as much as possible but longer delays sometimes taken do more harm than good.

Now, in these people who are very ill, theoretically the Miller-Abbott tube is awfully good, but I have had a lot of difficulty in getting that tube out of the stomach. They certainly can't be controlled adequately unless taken to the x-ray room, and most of these people are too sick to be transferred all around, and the delay attached to the use of the tube often does much more harm than good. There are cases where it is extremely useful and very safe to use; those are cases who are obstructed in the almost immediate post-operatives period where you can feel almost certain that the obstruction is due to inflammatory reaction and not to a band of adhesions that might strangulate as well as obstruct.

Some surgeons feel very confident in differentiating between the obstructed bowel and strangulated bowel. Well, I grant that in a large number of them you can do it, but I think there is quite a sizeable number of cases where it is almost impossible to differentiate between a bowel that is simply blocked and one that is strangulated, and if you delay on a strangulated bowel it is going to become gangrenous bowel and make a very much more serious situation.

Now I would like to say a word about aspiration of the bowel. There has been a tendency for a good many years against opening the bowel in the face of obstruction. There have been several excellent studies on the circulation of the bowel published recently, particularly by Johnson and his co-workers in Detroit, and they show very beautifully that intra-luminal pressure so compresses the veins in the wall of the bowel as to be one of the factors in the large effusion of fluid into the lumen, and, in addition to that, the circulation is so impaired that the bowel doesn't come back; so in these advanced cases where the bowel has a sort of bluish or greenish color and you know that the wall, while probably intact at that time, is at least threatened and the function of the bowel is destroyed, by putting a suction tip—which is usually about eight inches long—into that bowel, you can aspirate a very large quantity of fluid, and it is amazing to see how quickly the condition of the bowel improves. In one case we got 5,000 c.c. out of the small intestine. If you have an eight-inch suction type, by threading the bowel on it carefully you can empty about eight feet. Then, if that isn't enough, you can turn it around and aspirate eight feet in the other direction, so by emptying this bowel you restore the circulation and restore its function and avoid one of the dangers, which is continual loss of fluid into this damaged bowel, which might occur otherwise.

I do not believe there is any real danger of peritonitis in that. You don't get peritonitis from any small spillage into it. All of you have seen how a perforated appendix does if you get it early. Danger of infection is from a continual spillage like a perforated gall bladder or an appendix that isn't walled off, but that small amount you get from opening bowel isn't anything, and in addition to that we have the use of all the antibiotics.

Finally, I would like to make one observation on constriction of the colon. When you have an obstructed colon and a competent ileocecal valve you can get an enormous extension of the right colon and in the face of this distention decompression is the sound thing to do, but what I would like to mention is that if the obstruction is not of very long standing, if your distention is not too great and your lesion is in a mobile section of the bowel like the transverse colon or sigmoid, it is almost as easy to do your radical operation for removal of the tumor and then follow the Rankin procedure which he calls obstructive resection. It is just about as easy to do that as to do a transverse colostomy. A transverse colostomy sounds very easy, but if you have a fat patient it may be very difficult and the proximal end can be opened as soon as you can open your transverse colostomy.

I am sure all of you will agree that cecostomies of all kinds are unsatisfactory operations.

Dr. Putney reported one case where the mass in the sigmoid was inflamed; he resected the mass, exteriorized the proximal end and closed the distal end. I have done this several times when the lower end was too short for exteriorization and the condition of the bowel not suitable for immediate anastomosis. The continuity can be re-established at a later operation. I am sure that immediate end to end suture should only be done when the bowel ends are in prime condition.

THE TREATMENT OF SPONTANEOUS SUBARACHNOID HEMORRHAGE*

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Richmond, Virginia.

Subarachnoid hemorrhage had been noted as a post-mortem finding for many decades but it was not until the publication of Symond's classic paper in 1923 that much clinical interest was aroused. Numerous studies were then made but diagnosis rather than treatment was stressed. One important result of these investigations was the recognition of the extremely high mortality accompanying subarachnoid hemorrhage. More recent and comprehensive reviews, such as those of Richardson and Hyland, Hamby, Magee and Wolf, Goodell and Wolff have also emphasized the extreme gravity of this condition and have contributed data concerning the ultimate prognosis.

Available statistics indicate that approximately 35 to 50 per cent of patients die in their first episode of subarachnoid hemorrhage and another 10 to 25 per cent die in recurring attacks during the subsequent four weeks. An additional 5 per cent succumb during the first year after the initial hemorrhage, and, indeed, the untreated patient can never be free of the danger of a recurrence. Such recurrent hemorrhages have been reported as long as twenty-seven years after the original episode.

CAUSE

The possible causes of subarachnoid hemorrhage are legion but the most common causes are hypertension, aneurysms and arteriovenous malformations. This study does not include traumatic subarachnoid hemorrhage and is confined to the so-called spontaneous hemorrhage.

The precise cause of intracranial bleeding in hypertension is not yet perfectly understood. Apparently the initial lesions are small ring hemorrhages around the capillaries and these small areas sometimes coalesce to form large hemorrhages. In other instances the small peri-capillary hemorrhages weaken the supporting structures and permit large hemorrhages. It has also been noted that many cerebral hemorrhages seem to occur around veins. In most cases of hypertensive subarachnoid hemorrhage the original bleeding has been into the brain substance with secondary leakage into the subarachnoid space.

That aneurysms are frequent causes of subarachnoid hemorrhage cannot be doubted. Dandy, Hamby, Richardson and Hyland and many others have noted the frequency of these lesions and have stressed that fact that detailed search at the autopsy table will disclose many aneurysms that will not be seen in routine studies. The etiology of these aneurysms has received extensive study. A few of these lesions are undoubtedly mycotic and syphilis may be a causative agent. Many of the tortuous dilatations are due to arteriosclerosis but the vast majority of aneurysms are congenital in origin. Forbes postulated that aneurysms occur because of defects at the points of bifurcation of the arteries but the occurrence of many aneurysms at points distant from any branchings makes this theory untenable. The detailed study of Bremer and particularly the exhaustive work of Padget on the development of the cranial arteries by reorganization of capillary beds strongly indicate that aneurysms occur where embryonic vessels fail to completely resolve.

SYMPTOMS AND SIGNS

The symptoms of subarachnoid hemorrhage are well known and need be only briefly summarized. The onset is usually sudden and explosive, being initiated by headache and then unconsciousness. The neck is stiff and Kernig's sign is positive. The final proof, of course, is the obtaining of bloody spinal fluid. Frequently there are no localizing signs but sometimes there are such signs, e.g., hemiparesis, cranial nerve palsies (particularly 3rd nerve palsies). In the vast majority of patients there are no prodromal symptoms to indicate the presence of an aneurysm. However, localized pain over an eye and 3rd nerve palsies have been noted for weeks before the explosive onset of subarachnoid hemorrhage in some cases.

TREATMENT

A review of the literature and close observation of a considerable number of patients with subarachnoid hemorrhage has led to some conclusions as to evaluation and treatment of such patients. Each case must

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*Paper presented before the Richmond Academy of Medicine.

be considered individually and only a broad policy can be outlined. As a basic premise, it must be understood that this syndrome carries a very bad prognosis, no matter what treatment is employed. Any condition carrying a mortality rate of approximately 50 per cent is a real challenge to therapeutic endeavors. However, whereas approximately 50 out of 100 of these patients die, the fact must not be lost sight of that the other 50 will survive. The balance in all of these patients is precarious and everything done to them must be of real therapeutic value. Ill-advised surgical procedures will certainly increase the mortality rate. Although conservative or expectant treatment is no treatment at all, it seems preferable to utilize no treatment rather than poor treatment. Ideas on the proper treatment of cases with subarachnoid hemorrhage are subject to change as new evidence is presented but for the present an apparently rational scheme will be outlined.

No patients are subjected to any surgical procedure during the acute phase of subarachnoid bleeding. A diagnostic lumbar puncture is performed but repeated spinal drainage is not employed as it is not thought to be of any therapeutic value. The available literature mentions that the dangers of spinal drainage have been over-emphasized but it gives no real evidence that such treatment is at all effective. In the absence of such evidence it is felt that repeated spinal punctures should not be done. The patients are separated then into two groups as to treatment: the hypertensive and the normotensive. The elderly hypertensives for the most part are treated by medical measures directed at their hypertension. Although some of these patients may have aneurysms, it is most probable that their hemorrhage is on a hypertensive, arteriosclerotic basis. It does not seem logical to attack an isolated outpost of a generalized disease process in an ill patient. These cases are given large doses of rutin in an attempt to decrease the capillary permeability but otherwise are kept on a coronary regimen. Middle aged hypertensive patients are treated more vigorously but again the emphasis is on the hypertensive state in the absence of signs indicating a lesion at the circle of Willis. Two of these patients have had lumbodorsal sympathectomies and one such case is here reported.

Case Report: A 45-year-old white male was driving his car when he noted sudden onset of severe occipital pain. He drove several miles and the pain became more severe. He was admitted to a hospital on January 27, 1948, where his neck was found to be very stiff and Kernig's sign was positive. Neurological examination was entirely negative. Past history revealed that he was a known hypertensive of at least ten years standing. Blood pressure at time of admission was 190/115. Lumbar puncture revealed blood-tinged fluid. Kidney, cardiac and sedation tests were satisfactory and thoraco-lumbar sympathectomy was performed in two stages on March 10, 1948, and March 19, 1948. The patient has had no recurrence of subarachnoid hemorrhage and is working at his normal occupation.

Young hypertensive patients who do not have an obvious cause for the hypertension, such as coarctation of the aorta, are investigated for an intracranial aneurysm as later outlined, disregarding the hypertension for the moment. If no aneurysm or vascular malformation is discovered, the emphasis is again shifted to the hypertension.

The normotensive patients who recover from the original hemorrhage are considered as candidates for further investigation. Such further studies are carried out as soon as the patients' condition permits. During the acute phase nothing of a surgical nature is contemplated even if localizing signs are present. If any surgical procedure is ever carried out on these acute cases it should be an intracranial attack on the bleeding point. Ligation of the internal carotid artery during the acute phase of hemorrhage is apparently of no value. As soon as the patient responds arteriography is performed, whether localizing signs are present or not.

It is mandatory to try carotid compression to see if the patient can stand some curtailment of the cerebral circulation. If an aneurysm has been demonstrated and the patient cannot stand carotid compression, the internal carotid artery is partially ligated over a fascial band. Later a direct attack may be made on the aneurysm. If carotid compression produces no symptoms an intracranial approach is frequently made. If the aneurysm has a sac that can be obliterated a clip is placed on the neck of the sac and nothing else is done as is illustrated by the following case.

Case Report: A 33-year-old white male was admitted to the hospital on June 21, 1948, because of inability to raise his right eyelid. Three months before he had noted drooping of the right lid but this

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symptom disappeared within two weeks. Three weeks before admission he again noted inability to raise the right eyelid and this symptom persisted. Neurological examination revealed a complete right third nerve paralysis (inability to raise the right lid, eye held in external rotation and pupil dilated). Blood pressure was 130/80. Lumbar puncture revealed faintly xanthochromic fluid. Examination was otherwise entirely negative. Arteriography on June 24, 1948, revealed a berry aneurysm on the internal carotid artery (Fig. 1). On June 28, 1948

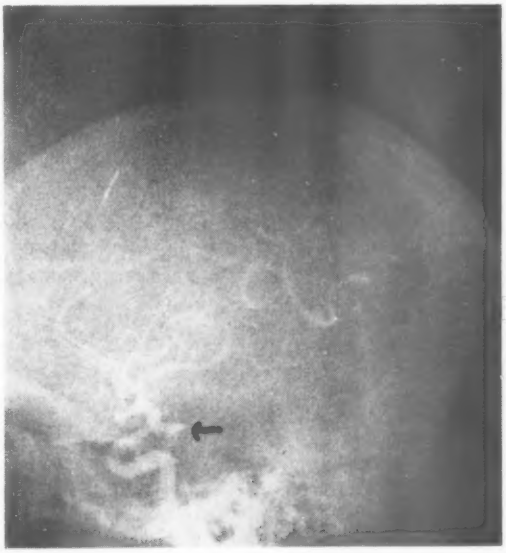

Fig. 1.

a right frontal craniotomy was performed and it was possible to put a clip on the neck of the aneurysm. The third nerve paralysis still persisted at time of discharge on July 6, 1948. The patient reported on September 8, 1948, that the third nerve paralysis had completely disappeared. He has continued well to present date.

If the aneurysm is on the carotid artery and does not have a sac that can be safely obliterated, an attempt should be made to trap the aneurysm between an intracranial clip and a ligature in the neck, as this will cure the aneurysm. On such occasions every attempt should be made to place the clip proximal to the posterior communicating artery in order to save the circulation provided by this vessel.

Arteriography may reveal an arterial lesion far removed from the base of the brain. In such instances it is well to attack the lesion directly as is illustrated by the following case.

Case Report: A 6-year-old white girl was admitted to the hospital on June 16, 1949, because of paralysis of the left side of the body. She had been perfectly well until two hours before admission when she suddenly complained of severe pain in the right side of her head. She soon developed a left hemiparesis and was extremely lethargic. Examination revealed only a left hemiparesis and stiffness of the neck. Blood pressure was 110/20. Lumbar puncture showed grossly bloody fluid. The patient slowly regained some function in the left arm and leg but developed bilateral papilledema. On July 1, 1949, she again suddenly became comatose and lumbar puncture revealed grossly bloody fluid. Patient gradually improved and arteriogram on July 6, 1949, revealed an aneurysm arising from the right callosomarginal artery (Fig. 2). On July 11, 1949, a large

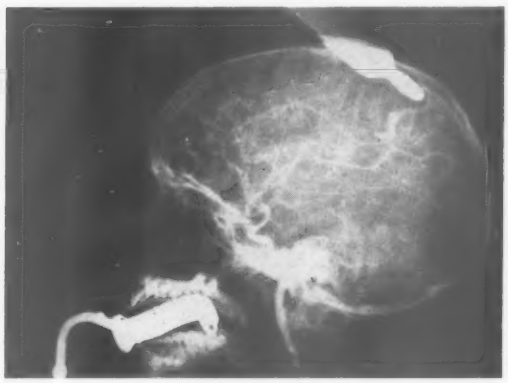

Fig. 2.

hemorrhage was evacuated from the right parietal lobe and the calloso-marginal artery was clipped. The patient was discharged improved on July 22, 1949. She has continued well and has regained almost normal strength so that she now takes dancing lessons.

Arteriography may disclose the presence of a vascular malformation. In most instances these cases have histories of many small episodes of subarachnoid hemorrhage rather than the massive bleeding commonly seen in arterial aneurysms. When these lesions are small and in an accessible area they can be removed. Usually, however, they are large and attempts at surgical removal are fraught with peril as to life and possible resultant neurologic deficit.

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The proper treatment in such cases is ligation of the internal carotid artery, thereby decreasing the systolic thrust. Such a procedure frequently prevents recurrent hemorrhages as illustrated by the following case.

Case Report: A 25-year-old white female was admitted to the hospital on October 2, 1949, because of severe headache. Twelve years before admission she had sudden onset of headache and was unconscious for four days. Three weeks before admission she suddenly developed a severe headache and lost consciousness for a short time. Lumbar puncture revealed bloody fluid. One week later she again developed severe head pain and again had bloody spinal fluid. At time of admission the patient was very lethargic and the neck was rigid. There were no localizing neurological signs. Bilateral carotid angiography on October 5, 1949, showed a vascular malformation in the left parietal region (Fig. 3). On

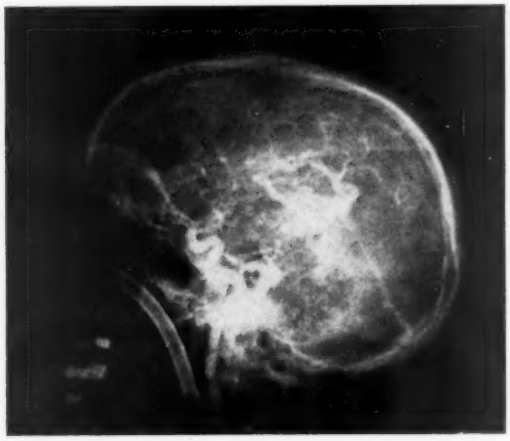

Fig. 3.

October 7, the left internal carotid artery was occluded in the neck. The patient's general condition rapidly improved and she was discharged on October 14, 1949. She has continued to be perfectly well with no complaints.

One must always be aware of the danger of any surgical attack on aneurysms. Even common carotid ligation carries some risk, as has been amply demonstrated by Pilz, Dandy and others. Attack on the lesion should always be the method of choice, as only by such direct attack can aneurysm be said to be definitely cured, but sometimes such a direct attack is not feasible. In some cases of carotid aneurysms the ligation of the internal carotid alone may be the only indicated treatment.

Efforts to reduce the prohibitive mortality of subarachnoid hemorrhage should be concentrated on two periods. The first of these important periods is before the hemorrhage actually occurs. Thus the hypertensive should receive thorough treatment, whether it be medical or surgical. The patient with a third nerve paralysis (ptosis of eyelid, dilated pupil, external rotation of eye) must have a presumptive diagnosis of aneurysm, particularly if there is also pain in the affected eye. If an attack is made on the aneurysm at this time the mortality will be low. As most aneurysms evidence no prodromal symptoms, the cases that can thus be treated before hemorrhage are few in number. The second phase deserving emphasis is the period when the patient rallies from the initial hemorrhage. Unfortunately present methods offer little benefit during the period of acute bleeding. However, efforts to prevent a second hemorrhage will greatly reduce the total mortality.

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SUBPHRENIC VAGOTOMY VERSUS GASTRECTOMY*

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There are so many ramifications to a discussion of this kind that I feel constrained to limit myself to one phase of the subject, that is, a comparison of the results obtained by elective surgery on complicated duodenal ulcer, using subdiaphragmatic vagotomy in one series and subtotal gastrectomy in the other. All the vagotomies have been accompanied by an operation in which a new stoma was formed—usually a gastrojejunostomy. A majority of the gastrectomies have been posterior Hofmeister operations. The indications for surgery have been intractable pain from peptic ulcer, failure of medical treatment after prolonged trial, pyloric obstruction, recurrence after previous perforations or repeated massive hemorrhages. The rationale of any operative procedure on the stomach with intractable duodenal ulcer is to reduce the acid content of the stomach either by removal of the acid-bearing portion, by side-tracking operation or by section of the nerves controlling the secretory cells. The physiological basis for this is largely empirical but has some foundation in experimentation as well as actual experience.

The history of vagotomy is a long and interesting one. As long ago as 1814, Benjamin Brodie[1] performed bilateral section of the vagus nerves in dogs and noted a decrease in gastric acid secretion, increase in mucus and dilatation of the stomach. Several other investigators since then have made tentative experimentation on this aspect of gastric surgery, but not until Pavlov began his experiments in 1910, was the subject fully explored.

Exner[1] in 1911 performed subdiaphragmatic vagotomy for gastric crises and later added gastroenterostomy. Latarjet[1] first reported on this procedure for ulcer in 1922. He and his associates performed 59 cases with excellent results but the procedure did not gain wide acceptance, probably because of the fear that vagotomy might have serious after-effects on the other viscera supplied by these nerves. There are several excellent historical surveys of vagotomy and I will not attempt to go into the history of the subject at this time. Before 1920, gastroenterostomy alone, or pyloroplasty, was frequently performed for duodenal ulcer,[2] but the frequency of gastrojejunal ulcers, and the recurrence of the duodenal ulcer, caused a gradual loss of favor for these procedures. After this date, gastrectomy received increasing attention and was performed with much greater regularity and success until the 1940's. In 1943, after years of careful research, Dragstedt presented a series of cases operated on by transthoracic vagotomy. Later he changed the technique and performed subdiaphragmatic vagotomies, frequently accompanied by gastroenterostomy. The purpose of this is clear; it gave a much better opportunity for abdominal visualization and exploration of the lesion in the stomach.

The rationale of vagotomy is founded on the belief that peptic ulcers develop in an increased acid medium. Dragstedt's[3] opinion is that the increased secretion occurs chiefly at night when the stomach is empty. This phase of gastric secretion is governed largely by neurogenic control. It has been definitely shown that patients with duodenal ulcer will have a markedly increased volume, as well as an increase in free hydrochloric acid during the period of rest. Section of the vagus nerves blocks the cephalic phase, and by experiment and actual practice has definitely shown itself capable of decreasing both total secretion and free hydrochloric acid. The Hollander intravenous insulin test, advocated by many to determine the completeness of vagotomy, in our series of cases has not been completely reliable. There are many instances in which the ulcer has healed both from the patient's subjective and objective examination and from laboratory and x-ray procedures, who still have a positive insulin test. There are others who have a negative insulin test with persistence of symptoms.

The basis of subtotal gastrectomy is also to reduce the acid content and gastric secretion volume, but is done by mechanically removing the acid bearing portion of the stomach. An attempt is made to resect

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about 1" of duodenum and ¾ of the stomach including most of the lesser curvature. The results are undoubtedly effective in reducing the acid, for nearly every case has a 0 free acid and a low total acid. The dreaded complications of this procedure, however, are: (1) Stomal edema or obstruction, (2) Post-operative hemorrhage and (3) Gastro-jejunal ulcer. The results of the two series of cases are classified under the following headings: (1) Excellent, (2) Satisfactory, (3) Improved and (4) Failure. The factors taken into consideration in the evaluation of the results are numerous and include both subjective and objective findings. The patient's own evaluation of his results is given a great deal of weight. The laboratory and x-ray findings are considered, and are naturally of primary importance. Forty-nine cases of duodenal ulcer were treated by vagotomy accompanied by a gastrojejunostomy, or, as in two cases, by gastrectomy also, and 44 cases by subtotal gastrectomy, in most cases using a posterior Hofmeister procedure with a short loop. It will be seen from a tabulation of the results that the outcome of the vagotomies is excellent in 13 or 26½ per cent of the cases; satisfactory in 34.7 per cent; improved in 32.6 per cent, and failure in 6.1 per cent. The total of excellent and satisfactory combined is 61.2 per cent. In the gastrectomy series there were 24 excellent results, or 54.5 per cent; satisfactory in 36.3 per cent; improved in 9.1 per cent and no absolute failures. The total of excellent plus satisfactory results combined is 90.8 per cent as compared with 61.2 per cent in the vagotomies. There were three deaths in the vagotomy series; however, only one of these could be ascribed to a recurrence or complication and this apparently was a hemorrhage. Of the other two cases, one died of an unrelated uremia and the other from an accident. None at all occurred in the gastrectomy series. However, it must be admitted that most of these latter cases have a shorter follow-up period. The results of these two series are very similar to those obtained by other authors with certain exceptions. Collins,[4] of the Cleveland Clinic, in July, 1950, found that better results were obtained at this clinic by the use of vagotomy plus gastroenterostomy. The same opinion is advanced by George Crile, Jr.,[5] at the same clinic. Gray and Williams,[6] in 1949, believe that partial gastrectomy is the preferable procedure. Beal and Dineen[7] obtained 90 per cent satisfactory results with gastrectomy, but had 17.4 per cent failures with vagotomy. St. John,[8] in 1948, and Glenn and Harrison,[9] in 1950, also favor gastric resection for treatment of duodenal ulcer. Among others who state

TABLE 1

	Subphrenic Vagotomies		Subtotal Gastrectomies	
Total	49		44	
Excellent	13	26.5%	24	54.5%
Satisfactory	17	34.7%	16	36.3%
Excellent plus Satisfactory	30	61.2%	40	90.9%
Improved	16	32.6%	4	9.1%
Failure	3	6.1%	0	0.0%

that gastrectomy is their operation of choice are, G. G. Miller[10] in the *British Journal of Surgery* in 1950, and Walters[11] of Mayo Clinic. Dragstedt still favors vagotomy in the treatment of the average ulcer of the duodenum, as does Grimson, of Duke. In evaluating the results of our series, personal contact, examination, and laboratory, plus radiological findings are given the greatest weight. However, later information sometimes was obtained by letter and in these cases, results were considered on the basis of questions asked. It is necessary, however, in questionnaires to know something of the personality of the patient. Frequently, the answers tend to be more pessimistic than those given to the same questions when delivered personally.

From the results in the two series, it is quite evident that this survey shows definitely more favorable results in the subtotal gastrectomies. The vagotomized patients not only have more post-operative gastrointestinal symptoms, but have much more frequent development of additional complaints, related apparently to the effect of altering the vegetative nervous system. Among the complicating factors of vagotomy are nervousness, vertigo, occasional diarrhea, weakness, sweating, epigastric pain, vomiting and eructation. The gastrectomy patients occasionally, but not commonly, develop nausea and vomiting, dumping syndrome with its weakness, nausea and sweating, and a few other lesser complications. Major complications such as definite recurrence of the duodenal ulcer has not been proven in the vagotomy series, but at least two cases had later hemorrhages which may have been due to gastrojejunal ulcers. None of the gastrectomies have developed definite stomal ulcers and none have been complete failures.

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The gastric acidity has shown a tendency to rise to normal levels again in one to two years after vagotomy, probably due to the development of an automatic control by the submucous plexus rather than regeneration of the vagus nerves. VanZant[2] has shown that vagotomized dogs show no regeneration of the vagus fibers, but have developed normal acid volume and percentage after several years. The cases in which vagotomy was done and who have developed an increased acid later on, have not necessarily developed ulcer symptoms again, however.

In conclusion, it is of interest to note that the initial fervor with which the operation of subdiaphragmatic vagotomy was met has now settled down to a more practical attitude of case selection, using this operation more often for cases in which it would be extremely difficult, or dangerous, to remove the duodenal ulcer and for marginal ulcers. The average case of duodenal ulcer requiring surgery should, in our opinion, be treated by subtotal gastrectomy. Finally, I am convinced that any type of operative approach to duodenal ulcer, except pyloric obstruction perforation or near fatal hemorrhage, is, despite frequent brilliant results, but a temporary expedient, which will some day give way to a less brutal and more fundamental form of therapy. What this will be is not within my power and I daresay anyone else's power to say, but it may well be in an exploitation of a more comprehensive knowledge of the real cause of ulcers and a more superior control over body chemistry, hormonal and enzymatic balance, coupled with neuropsychiatric treatment. This may be strange language from a surgeon, but someday the difficult riddle of peptic ulcers will be solved, and I believe that surgery then will take second place in its treatment.

ADDENDA

Since completion of this article, two cases of subdiaphragmatic vagotomy, both of whom have been followed from time to time and both of whom were operated on in 1947—one in November and one in July, have developed further trouble, and must be classified as failures. Both have had gastric hemorrhages and one shows definite gastrojejunal ulcers. A subtotal gastrectomy will be the solution in both cases.

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DISCUSSION

DR. HUGH H. TROUT, JR., Roanoke: I should like to congratulate Dr. Rennie on his timely paper and also for his good results. The early results, as reported by Dragstedt, who put out the first large series, were extremely excellent; and yet, when we look back, the results during the first six months or year when gastro-enterostomy came into general use were even better than have ever been reported for vagotomy or subtotal gastric resection. But the trouble was that sufficient time had not elapsed for the development of jejunal or stomal ulcers in the early gastro-enterostomy cases, and the late ulcers were found to be much more frequent than the early results indicated. The same thing is true also of vagotomy. It was greeted with great enthusiasm; yet, particularly as shown in Dr. Rennie's paper, the results are not as good as those with subtotal gastric resection. Subtotal gastric resection, of the three operations, has been subjected to the longest period of trial and has held up. I agree with Dr. Rennie that in many ways it is not physiological and that perhaps some better treatment will come, but in general it is my feeling that that is the treatment of choice at present.

While Crile and Dragstedt and a few other surgeons still feel that many ulcers should be treated with vagotomy, I believe that the vast majority of the surgeons in America now feel that the main indication, as Walters has pointed out in a large series at the Mayo Clinic, is for stomal ulcers following a gastric resection. Cer-

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tainly the usual duodenal ulcer should be treated with resection; that is, provided it is a case for surgery. If the ulcer is extremely difficult to remove, as certain penetrating ulcers far down in the duodenum are, it is my personal opinion that an exclusion operation will give better results than vagotomy, even though the ulcer is not removed. A stomal ulcer following a gastro-enterostomy is best treated by excision of the ulcer and a subtotal gastric resection.

Recently Grimson and his associates have reported on banthine, which is an amine compound causing autonomic blocks of the nervous system. By the use of this compound a so-called "medical" vagotomy can be produced. They first reported one hundred cases, with unusually good results. In my experience and that of my associates our results have been encouraging but nothing as striking as reported from the Duke group. Certainly, however, this gives us food for thought; and, while banthine cannot be considered as effective an autonomic block as a vagotomy, still it could perhaps be relatively comparable and could be tried. Now, if banthine is going to be effective in a patient there is no reason why it cannot be continued and vagotomy perhaps not be necessary. If, on the other hand, banthine is not effective in a certain patient, one would certainly be rather skeptical as to the efficiency of performing a vagotomy in that patient.

I believe that vagotomy will eventually hold a place in gastric surgery, perhaps in stomal ulcers following resections; but the place is becoming smaller, and I think that Dr. Rennie has brought that out very clearly.

DR. RENNIE, closing the discussion: I wish to thank Dr. Trout for his excellent discussion and to add that it is very difficult to evaluate the results in these patients, as so many of them are cases in which you cannot ascertain the degree of subjective improvement, because of the strong pension influence. You have to take so many of the answers with a grain of salt and do the best you can.

Reversible Bifocals.

A new type of eye glasses which allows the bifocal portion to be moved out of the way when not needed is described by Dr. David E. Rolf of Cleveland in the April *Archives of Ophthalmology,* published by the American Medical Association.

According to Dr. Rolf, the lenses of the glasses may be rotated, or turned upside down, by a unique method of attachment, so that the bifocal part is on top instead of on the bottom. In his opinion this reversible lens arrangement has certain advantages. "Its flexibility will save many patients an extra pair of glasses. It offers a possible solution to the long-standing problem of the patient who cannot accustom himself to walking while wearing bifocals in their usual position. It also permits the bifocal segment to be placed upward and out of the way during all or part of his walking activities. It would also seem applicable for patients engaged in occupations with unusual visual demands. Patients in these categories include barbers, switchboard operators, painters and paper hangers, as well as most people employed in the building trades, where sure footing is a necessity."

Dr. Rolf believes that the mechanics of changing the position of the lenses are sufficiently simple so that most persons are able to master them easily. There are no limitations regarding size, shape or type of bifocals or trifocals that can be used and the technic of prescribing and fitting these lenses is not difficult.

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INCIDENCE OF INTRACRANIAL NEOPLASMS IN VIRGINIA

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During the period 1920 to 1950 at the Medical College of Virginia Hospitals, 249 intracranial neoplasms have come to the autopsy service of the pathology department. These lesions, which were recorded during the examination of 6,300 consecutive post mortems in this interval, constitute approximately a 4 per cent over-all incidence. Since most of these cases were from Richmond and the surrounding region, we feel that the following statistics probably represent average figures for the state of Virginia and possibly for any of the Southern states.

FIGURE 1

TABLE OF COMPARISON OF INCIDENCE OF INTRACRANIAL NEOPLASMS

Classification of Neoplasm	*Anderson*	*Courville*	*Pautler/Apperly*
1. Chordoma	1%	?	0% (0)
2. Glioma (Total)	44%	38.8%	50% (124)
A. Astrocytoma	13%	5.6%	8% (20)
B. Glioblastoma multiforme	11%	22.8%	28% (69)
C. Medulloblastoma	5%	3.9%	6% (15)
D. Ependymoma	4%	2.8%	2% (5)
E. Oligodendrogliocytoma	3%	0.6%	0% (0)
F. Astroblastoma	2%	0.8%	1% (3)
G. Spongioblastoma unipolare	1%	?	1% (3)
H. Others	5%	2.2%	3% (9)
3. Neurinoma	10%	3.9%	4% (11)
4. Meningioma	15%	10.7%	9% (22)
5. Mesodermal (Total)	4%	1.4%	5% (13)
A. Vascular	3%	1.4%	4% (11)
B. Stromal	1%	?	1% (2)
6. Mixed (Total)	5%	1.1% (cong.)	3% (8)
A. Dermoid, epidermoid, etc.	1%	?	1% (3)
B. Craniopharyngioma	4%	?	2% (5)
7. Hypophyseal	16%	5%	9% (23)
8. Metastatic	3%	18%	16% (37)
9. Granuloma	2%	21.4%	4% (11)
		0.2% (misc.)	

The accompanying table with its classification and percentages (see Fig. 1) was obtained from Anderson[1]. His figures undoubtedly represent averages for the northern states and possibly for the nation as a whole. They are based on over 2,000 cases (including those of Cushing) and consist mainly of surgical specimens, although many of the tumors were verified at autopsy. Also included in this table are the figures of 355 intracranial neoplasms found in a series of consecutive autopsies by Courville[2].

In the comparison of Anderson's and Courville's findings with ours, both the similarities and the differences are of interest and worthy of comment. First of all, the gliomas in general constituted 124 cases, or 50 per cent in our series, which is slightly higher than the other two figures. We found the commonest glioma to be the glioblastoma multiforme with 69 cases (28 per cent), which is almost three times more frequent than Anderson's 11 per cent but right in line with the 22.8 per cent of Courville. Anderson, as well as other texts, lists astrocytoma in first position. The figures for the astrocytoma, medulloblastoma, ependymoma, astroblastoma and spongioblastoma are either identical with or quite similar to

We are indebted to Dr. C. C. Coleman and the Department of Neurosurgery through which most, if not all, of this material came.

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the others. It is to be noted that no oligodendrogliocytomas have come to autopsy at this hospital.

As for the other types of neoplasia, we gathered 23 cases (9 per cent) of hypophyseal tumors, of which all but one were adenomas and 50 per cent of these were of the chromophobic type. As for the meningiomas, neurinomas, mesodermal, mixed and metastatic types, all figures are within reasonable limits. The low percentage given by Anderson for the metastatic variety is understandable in view of the fact that his series is based primarily on surgical material. Of our series, about two-thirds, or 26 cases of the metastatic type, were due to carcinoma. According to the percentages of Anderson (2 per cent) and ourselves (4 per cent) for the granulomas, it appears that the 21.4 per cent of Courville is unreasonably high.

Although the collection of tumors presented is not large and the possibility of misdiagnosis must be considered, we feel that the comparison of the Virginia figures with others around the country is worth while.

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Nerve Surgery Helps Paraplegics in Convalescent Period

Nerve surgery on some paraplegics (persons paralyzed in the lower part of the body) in a convalescent stage appears to be justified as a result of experiences at the Van Nuys (Calif.) Veterans Administration Hospital, according to Dr. John D. French, associated with the hospital.

Dr. French, reporting on his observations of 500 paraplegics in May 19 J.A.M.A., explained that at first a paraplegic is under the care of a neurosurgeon. The neurosurgeon evaluates the extent of the injury and does what he can, surgically and otherwise, to facilitate the return of function where damage is not irreversible. Next comes a period of rehabilitation, often long and difficult, during which neurosurgery contributes little. Later, however, he pointed out, rehabilitation may stop short of the ultimate goal for the patient because of new neurosurgical problems which can be helped by a second operation. Seventy-seven of the 500 patients he reported fell into this category and were operated upon because their rehabilitation became static or regressive.

As an illustration, he cited the case of a 30 year old man who became paralyzed in the lower part of his body as a result of a shell fragment. He said:

"Rehabilitation was never satisfactory because the patient complained of pain in both legs. He was admitted to the hospital three years later, where he made no effort to enter into the usual activities, of rehabilitation apparently being confined to the discomfort. A chordotomy [cutting a part of the spinal cord which carries pain] was done two years later after which complete relief of pain was reported. The patient was subsequently discharged from the hospital and now indicates that he leads a normal, comfortable existence."

Because of these and other advances in rehabilitation of paraplegics in recent years, Dr. French said that the feeling of hopelessness that clouded the outlook of these patients has been largely dispelled. They can be taught to talk and walk and can be returned to usefulness, he added.

He estimated that at least one out of every five or six paraplegics requires neurosurgical intervention late in his rehabilitation period.

SOME CONSIDERATIONS OF THE PHYSIOLOGICAL EFFECTS OF CORTISONE AND ACTH IN MAN.*

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Kendall,[1] in reviewing the trials and tribulations connected with the development of compound E, states that in 1946, interest in the steroid hormones of the adrenal cortex had sunk to a very low level. Many investigators had concluded that no hormone of the adrenal cortex was of sufficient importance to merit additional investigation of its characteristics, and more than one clinician lost all hope of these substances ever achieving any importance in the practice of medicine. This situation was suddenly reversed in 1949 when the first publication on the effect of cortisone in rheumatoid arthritis was published.[2] We now know that the adrenocorticotropic hormone of the pituitary and 17 hydroxy—11 dehydro-corticosterone or cortisone are powerful hormonal substances which have far reaching effects in man. It is likely that all of the body tissues are affected when these compounds are administered. They not only effect beneficial results in certain diseased states, but they also bring about a series of physiological and pharmacological alterations which are not always easily separated. The mechanisms by which these agents produce these therapeutic alterations remain obscure, but it is certain that they result from an artificial state of hormonal excess, for, with a few exceptions such as Addison's disease and panhypopituitarism, no hormone deficiency has been demonstrated in those diseases which are benefited by their administration. For these reasons, it seems important to study the physiological effects of cortisone and ACTH in man, for their safe and effective therapeutic employment depends on a thoroughgoing understanding of their physiological potentialities.

Inasmuch as these hormones are capable of inducing changes similar to those in Cushing's syndrome, it may be helpful to study the physiology of this condition. Cushing's syndrome may be produced by a functioning tumor of the adrenal cortex, a progesterone producing tumor of the ovary or by atrophic changes in the paired paraventricular nuclei of the hypothalamus.[3] Any of these primary causes can effect similar changes in the glandular hypophysis; that is, over-action of the eosinophile cells or degranulation of the basophilic cells. In the experimental dog, pathologic changes similar to those exhibited by persons with Cushing's syndrome can be produced by denervation of the neural hypophysis or by interruption of the afferent nerve pathways to the supraoptic and paraventricular hypothalamic nuclei. It has been shown that a Cushing-like syndrome can be produced by effecting a loss or a decrease in those factors of the neural hypophysis which are under the trophic influence of the paired paraventricular nuclei either by dividing the afferent fibers to these nuclei, or by dividing those issuing from these nuclei to innervate the hypophysis. On the other hand, it has also been shown that hyperfunctioning of these nuclei can effect changes in the hypophysis which are responsible for exophthalmic hyperthyroidism.[4] Normally there exists an antagonistic relationship between the hormones secreted by the inner two zones of the adrenal cortex and those hypophysial secretions to which the paraventricular nuclei are trophic. The metabolism of carbohydrate, fat and protein and sex gland development and activity are dependent on these hypophysial hormonal factors. When these factors are decreased, there is an increased secretion of the hormones of the adrenal cortex. Likewise, experimental evidence exists which indicates that those hormone factors of the neural hypophysis which are under the trophic influence of the supraoptic nuclei of the hypothalamus are entirely concerned with the regulation of salt and water balance. The secretion of desoxy-corticosterone or some similar steroid of the glomerular zone of the adrenal cortex is in balanced antagonistic relation with the antidiuretic principle of the pituitary.

As noted above, when there is a loss of those hypophysial hormones to which the paraventricular nuclei are trophic, there is increased hormonal activity of the two inner zones of the adrenal cortex. The increased activity is an expression both of increased secretion and augmented effectiveness. The effectiveness is increased by a loss of the normally antagonis-

*Read at the Augusta Medical Society, January 3, 1951.

tic hypophysial hormonal factors. This results in increased stimulation of hypophysial eosinophile activity, which, in turn, stimulates the adrenal cortex to hyperplasia and increased secretion. Therefore, there is established a self-sustaining cycle resulting is continued hyperfunction of the hypophysial eosinophile—adrenal cortical hormone complex. This results in a chain of characteristic symptoms such as development of increased appetite, obesity, decreased insulin response, hypertension of the diastolic type, cardiac, adrenal, renal and parathyroid hypertrophy, hypercholesterolemia and disturbance in potassium metabolism.

Polydypsia and polyuria are accounted for by neutralization of the antidiuretic hormone of the hypophysis by excess amounts of adrenal cortical hormone.[5] Obesity results from over-eating effected by polyphagia which is attributed to hyperfunction of hypophysial eosinophile adrenal cortical activity. This activity also interferes with the utilization of carbohydrate and apparently increases gluconeogenesis from protein. Therefore, there is additional carbohydrate for conversion into fat.

Degeneration of connective and elastic tissue in the arterial wall has been found in Cushing's syndrome.[3] Parathyroid hyperplasia and hyperfunction with demineralization of bone attributed to hypophysial basophile depression occurs in both Cushing's syndrome and renal dwarfism. It has been postulated that the calcium released by demineralization of bone is deposited in these degenerated elastic and connective tissue fibers which have been shown to be the ones where calcification occurs in atherosclerosis. What part the hypercholesterolemia of Cushing's syndrome plays in this picture is not known but arteriosclerosis is a constant development in the disease.

It has long been known that the cells of the renal tubule are under the trophic influence of the hypophysial eosinophile cells. These may stimulate the renal tubular epithelium to augmented excretion as measured by diodrast or para-aminohippurate. Renin when released by renal tubules in turn stimulates the hypophysial eosinophile adrenal cortex mechanism. This situation can be obtained in the experimental dog by wrapping one kidney with silk. With the development of hypertension, histological evidence of hyperplasia of the eosinophiles of the hypophysis becomes obvious. If the kidney is then unwrapped and normal circulation becomes re-established, tubular function returns to normal and the cytologic picture of the pituitary returns to its former state. Another possible explanation for the hypertension is through epinephrin and the adrenal medulla. Epinephrin not only causes renal afferent vasoconstriction with increased release of renin, but it is also now well known that it increases the secretion of adrenal cortical hormone[6] through the hypothalamic nuclei and eosinophiles of the hypophysis. These in turn apparently secrete a renotrophic hormone, as noted above. It also seems likely that as arteriosclerosis develops in all the arterioles, including those of the kidney, augmented renin release will occur in an effort on the part of the body to maintain adequate renal blood flow.

As this hypertension develops, the heart is stimulated to maintain the elevated pressure through the action of such mechanisms as angiotonin, the anterior pituitary and epinephrin and the adrenal medulla.

Colowick, Cori and Slein[7] have shown that the adrenal cortical hormone inhibits the utilization of carbohydrate by depressing insulin sensitivity. Heinbecker[8] has shown histological evidence of pancreatic islet cell depression in the experimental animal with the Cushing-like syndrome. Campbell and Best[9] and others have also demonstrated functional depression of islet tissue by the administration of the anterior pituitary hormone. Ham[10] has shown histological evidence of beta cell depression following administration of the hormone. Therefore, with this evidence in hand, it seems obvious that increases in hypophysial eosinophile adrenal cortical activity as a result of depression of the paired paraventricular nuclei are sufficient cause for the diabetes of Cushing's syndrome.

Osteoporosis is invariably found in Cushing's syndrome and is the result of parathyroid over-activity. This hyperfunction is accompanied by hyperplasia and is effected through degeneration of the hypothalamic nuclei with consequent depression of pituitary basophile activity.

From a clinical standpoint, it is convenient to divide the physiological activity of ACTH and cortisone into five groups: (1) effect on water and electrolyte metabolism; (2) effect on adrenal cortical function and structure; (3) effect on carbohydrate,

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protein and fat metabolism; (4) miscellaneous hormonal effects and (5) neuropsychiatric changes.

EFFECT ON WATER AND ELECTROLYTES

The effect of the adrenal cortical hormone on electrolyte and water balance is not clearly understood. Originally, it was thought that the chief effect of cortisone and allied hormones in this connection was the loss of salt and water from the body. In the adrenalectomized dog, retention of potassium and large losses of sodium with rapid elevation of plasma NPN are consistently found. These changes are easily reversed when adrenal cortical hormone is given. These effects do not invariably hold true when cortisone or ACTH is administered in man. In many circumstances they may cause a retention of salt and water, and frequently of sufficient degree to cause edema. This salt and water retention is not necessarily related to the prolonged administration of the drug or to excessively large doses. We have seen twelve pounds of water retention with periorbital and ankle edema develop after the administration of only 300 mgs. of cortisone, but usually it begins to appear only after 2 gms. or more of cortisone or 600 mgs. of ACTH have been given. In many instances excess salt and water will be excreted and edema will disappear in spite of the continued administration of the hormone. All of these effects have not been clearly elucidated. It has been suggested that one factor may be the depression of the endogenous production of corticoids by the administration of cortisone, but this certainly will not explain the salt and water retention which occurs when ACTH is given.

This sodium and water retention during the administration of cortisone or ACTH may create a hazard in the patient and usually does in those with cardiac complications. This is of particular importance in regard to rheumatoid arthritis, for a small percentage of these patients have mitral valve lesions similar to the mitral stenosis of rheumatic fever and Libman-Sachs disease. This lesion was first reported by the Mayo Clinic group in 1941, and, although it has not received very extensive attention in the literature, it is not an uncommon lesion in our experience. We have encountered one patient with rheumatoid arthritis and mitral disease who experienced severe cardiac decompensation with marked salt and water retention while under treatment with cortisone.

Losses of chloride and potassium with resulting alkalosis similar to that associated with Cushing's syndrome have been known to occur in patients receiving 200 mgs. of cortisone or 100 mgs. of ACTH daily. This situation is not exactly the same with the two hormones, inasmuch as there is also a lowering of plasma sodium when it develops as a result of the administration of ACTH. This does not occur during cortisone therapy. When the hormones are discontinued these electrolytes return to normal more slowly after cortisone than after ACTH.

Increased losses of potassium with a negative potassium balance invariably occur when either agent is administered in large doses. Initially, there apparently is a slight retention of potassium, but this rapidly gives way to increased losses with development of negative potassium balance. This potassium is derived from both intra and extracellular fluid compartments. The portion originating from within the cell is evidence of the catabolism of cellular protein which usually takes place when either hormone is administered.

EFFECTS ON ADRENAL CORTICAL FUNCTION AND STRUCTURE

It is probable that all known functions of the adrenal cortex are stimulated by the administration of ACTH. Actually, more compound F than cortisone is probably elaborated after ACTH administration. Morphologically there is both hypertrophy and hyperplasia of the adrenal cortex. On the other hand, when cortisone is administered, the effects on the cortex of the adrenal are generally opposite. Eventually atrophy takes place. This is noticeably seen in patients with Cushing's syndrome due to a hyperfunctioning adrenal cortical tumor on one side. The contralateral gland is usually hypoplastic and functionally inert. That atrophy and hypofunctioning of the adrenal cortex does occur, during and following cortisone therapy, is evidenced by diminished response to the Thorn test (fall in eosinophiles after ACTH administration), diminished excretion of 17-ketosteroids during and after cortisone administration, and by necropsy evidence of adrenal cortical atrophy. The glands show weight reduction and histologically there is depletion of lipoid material in the zona fasciculata while the zona glomerulosa is

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well preserved. This atrophy probably results from the suppression of pituitary adrenocortico tropic function which cortisone induces. There is, however, no reason to suspect that this alteration in function and structure is not reversible when the hormone is discontinued.

EFFECT ON CARBOHYDRATE, FAT AND PROTEIN METABOLISM

Carbohydrate: It seems obvious that the impairment of carbohydrate metabolism that one might expect from cortisone or ACTH administration seldom develops to an appreciable extent. This is probably due to the relatively small dose given as compared with that necessary to induce diabetes in the experimental animal. We have not seen diabetes develop in any patients under treatment with either of the hormones. We have given cortisone in the usual dosage to one patient without frank diabetes, but with definitely impaired tolerance to glucose, who showed no change in the glucose tolerance curve after four weeks of treatment consisting of a total of 3.1 gms. of cortisone. Normal glucose tolerance curves have been demonstrated after 90 and 160 days of cortisone administration. However, a temporary state of diabetes has been produced by both cortisone and ACTH. Conn, Lewis and Wheeler[11] report glycosuria due to lowered renal threshold for glucose. Frank diabetes with acidosis developing in a 17-year old boy with leukemia being treated with cortisone has been reported. McEwen[12] noted the development of diabetes in a 10-year old girl with Still's disease being treated with ACTH. Temporary exaggeration of pre-existing diabetes has also been reported by others.

Protein: With small doses of cortisone only small increases in nitrogen losses occur, but with larger doses (200 mgs. cortisone daily or 100 mgs. ACTH), there is a decided loss of nitrogen from the body which is due to increased protein catabolism. Pronounced creatinuria also occurs when larger doses are given, which also probably indicates increased protein breakdown, muscle protein in particular. The increased excretion of uric acid is consistent with the same interpretation. The development of cutaneous striae, the delay in wound healing and the inhibition of tumor growth which has been noted experimentally are all indications of the effects of these agents on protein metabolism.

Fat: The ways in which fat metabolism is influenced by cortisone and ACTH are not fully known, but, as has been noted elsewhere, fat stores are probably augmented by polyphagia, decreased utilization of carbohydrate and increased carbohydrate for conversion to fat by virtue of increased gluco neogenesis. Transient ketonuria in patients receiving 80 mgs. of cortisone daily has been reported by Perera in association with marked interference with carbohydrate metabolism. Abnormal distribution of fat consistently occurs with excess hormone administration. The trunk and neck become obese and the face rounded. Rounding of the face is usually associated with considerable weight gain. This change in facial contour is not due to edema, but to deposition of fat in the cheeks and it is usually the earliest and most easily noted sign of abnormal fat distribution.

MISCELLANEOUS HORMONE EFFECTS

Menstruation may be disturbed during the administration of either cortisone or ACTH. This usually manifests itself as amenorrhea or oligomenorrhea, and it would seem that menstrual function might be more easily disturbed in the young than in the mature female.

Mild hirsutism and acne occasionally develop in women which are effects usually associated with steroids exhibiting androgenic activity. More pronounced evidences of masculinization, such as deepening of the voice and enlargement of the clitoris, apparently have not been described. Exactly why these evidences of increased androgenic activity should occur is not clearly understood. It has been suggested that cortisone itself has weak androgenic properties or that it is metabolized to produce androgenic steroids. It is of interest to note that the 17-ketosteroids are not invariably the same qualitatively. Those produced in normal individuals appear to have dermatotrophic properties in the sense that they stimulate the growth of pubic and axillary hair and acne, but are not androgenic. Patients with adrenal cortical virilism, however, produce cortical hormones which prompt masculinization and nitrogen anabolism.[13]

NEUROPSYCHIATRIC CHANGES

Psychic effects are almost constantly seen during administration of these substances. Euphoria or mild mental stimulation almost invariably occur. Manic and depressive states and frank psychosis

have also been observed by others. In nearly all patients there is some alteration in mood. Hoefer and Glaser[14] report patients showing insomnia, irritability, bitterness and depression with well defined electroencephalographic changes. Hence, it is not likely that the sense of well being and euphoria seen in most patients is simply a result of surcease from discomfort. Thorn states that there are personality disorders in both hypo- and hyper-adrenal cortical function. It is interesting to note that the electroencephalographic abnormalities of Addison's disease can be corrected with cortisone. Hoagland has found that these changes in the adrenalectomized animal can also be corrected with Δ 5 pregnenalone.

SUMMARY

The adrenocorticotropic hormone of the hypophysis and cortisone are powerful hormonal substances with far flung physiological implications throughout the body tissues and systems. A thoroughgoing understanding of the potentialities of these hormones is an indispensable requirement for their secure and efficacious use.

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SYMPATHOLYTIC DRUGS IN PERIPHERAL VASCULAR DISEASE

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In 1945 Acheson and Moe[1] thoroughly investigated the effects of tetraethylammonium (a quaternary ammonium ion) in animals. They demonstrated that this drug blocked transmission of nerve impulses through the autonomic ganglia. It is believed that transmission of impulses from preganglionic to postganglionic fibers is mediated through acetylcholine liberation at the ganglion synapse. Tetraethylammonium acts by preventing the action of acetylcholine on the synapse of the post-ganglionic fibers. Hence, no impulse travels down these fibers. Tetraethylammonium chloride is known as etamon. The drug should be given intravenously or intramuscularly in 200 to 500 mgm. doses. Horwitz, Montgomery, Longaker and Sayen,[2] have demonstrated vasodilatation in peripheral blood vessels and moderate increase in cutaneous blood flow by its use. Etamon frequently causes alarming side reactions, such as, weakness, nausea, numbness, tachycardia, and a precipitate fall in blood pressure. At times it has proven an excellent testing device in the detection of pheochromocytoma, causing a sudden rise in blood pressure. The effect on hypertension in general is a lowering of the blood pressure. This property of the drug has been utilized in testing patients who have hypertension for splanchnic nerve resection. Our experience with etamon has been confined to hospital cases, as we do not believe that it is a safe drug to use in ambulatory patients. It is a popular testing device, but individuals with organic peripheral vascular disease have not shown any remarkable improvement by its use and those with gangrene have gone on to amputation.

In 1939, Hartmann and Isler[3] suggested that, in a group of phenyl substituted alkyl imidazolines, priscoline caused the greatest depression of blood pressure. It also causes vasodilatation in peripheral blood vessels, with slight increase in cutaneous blood flow.[2] Later, other investigators suggested that the action of priscoline was adrenolytic, preventing the liberation of epinephrine at the sympathetic endings in the smooth muscle of blood vessels, peripherally. Priscoline may be given orally or parenterally and, therefore, has a distinct advantage over etamon. There have been many studies in the foreign literature concerning its value. The investigations in this country by Grimson, Reardon, Marzoni and Hendrix,[4] also Rogers,[5] have caused enthusiastic acceptance by many clinicians.

Our experience with priscoline is based on the study of forty-two cases of peripheral vascular disease over the past two years. These cases were seen in the out-patient clinic and hospitals of the Medical College of Virginia. There were thirty-five patients with arteriosclerosis obliterans, five with thromboangiitis obliterans, and two with Raynaud's disease. The severe cases were those with varying degrees of gangrene, who were given 50 mgm. of priscoline parenterally twice daily in the hospital. In no instance was a limb saved from amputation by the use of the drug. The clinic and private cases were given from 100 to 200 mgm. of priscoline orally every day for a period of from three to eighteen months. They were followed with oscillometric studies and careful questioning as to intermittent claudication. There were no spectacular results and no improvement in the oscillometric readings. An occasional patient stated that he could walk a block further than before the use of the drug. Our best results were in ten private cases who discontinued smoking and followed all of our suggestions as to care of the extremities. These cases were considered mild and we thought they would have improved without the drug. Both of the cases of Raynaud's disease stated that episodes of cold fingers and numbness were lessened and they were happier while under the influence of the drug. Very few individuals could tolerate over 150 mgm. of priscoline orally a day. Their usual complaints were nausea, dizziness, and palpitation.

We attempted to evaluate the effect of priscoline on hypertension by the following experiment. Seventeen patients with hypertension were given 50 mgm. of priscoline intravenously. The blood pressure was taken before injection and every half hour for the

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next two hours. Our results showed no changes in ten cases, five cases had a rise of at least 10 mm. in systolic pressure, two had a drop of 20 mm. in systolic pressure. It has been suggested that either larger doses or prolonged use of the drug might be necessary to reduce blood pressure. We treated eight private cases with hypertension over a period of a year and none of them had any unusual changes in their blood pressure.

It is possible that both etamon and priscoline in large enough doses will produce general vasodilatation and dilate cutaneous blood vessels, but the sympatholytic drugs are not selective in their action, and general vasodilatation in other areas may tend to divert blood away from the skin. If there is a reduction of blood pressure due to vasodilatation, secondary vasoconstrictor reflexes tend to counteract the desired cutaneous vasodilatation. If other vascular beds are dilated, there may be an increase in cardiac output per minute, which is undesirable in cardiac disease, frequently coexisting in many patients with peripheral vascular disease. Our problem in peripheral vascular disease is to induce local vasodilatation to the affected extremity. This is best accomplished by local cutaneous nerve blocks or sympathetic nerve resection. Under the circumstances, we are not sure that the approach to treatment of peripheral vascular disease is with drugs that produce general vasodilatation. Our own clinical experience indicates that etamon and priscoline are of negligible value in the treatment of organic peripheral vascular disease.

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New Books

We give below some of the newer books received by the Tompkins-McCaw Library of the Medical College of Virginia, Richmond. These are available to our readers under usual library rules, by addressing the Library.

Alström—A study of epilepsy in its clinical, social and genetic aspects.
Army medical library—Classification. Medicine. 1st ed.
Bohm—Massage, its principles and technic.
Cecil and Loeb—A textbook of medicine. 8th ed.
Chobot—Pediatric allergy.
Current biography. Who's news and why. 1951.
Duncan—Diabetes mellitus.
Edsall, ed.—Enzymes and enzyme systems.
Greenberg—Antipyrine, a critical bibliographic review.
Himwich—Brain metabolism and cerebral disorders.
Hogan—Ocular toxoplasmosis.
Lutz—Richmond in World War II.
Public health service—Public health areas and hospital facilities.
Rosebury—Experimental air-borne infection.
Seymour—Rice, dietary controls and blood pressure.
Smith, H. W.—The kidney, structure and function in health and disease.
Strauss & Strauss—New ways to better meetings.
Veterans Administration—Conference on cortisone research. Symposium.
Winters, ed.—The collected papers of Adolf Meyer. Vol. 2, Psychiatry.

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MESENTERIC VASCULAR OCCLUSION WITH SUCCESSFUL RESECTION OF THE SMALL INTESTINE— REPORT OF A CASE.

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The recent report in the *Journal of the American Medical Association* of a case of mesenteric vascular occlusion with successful resection of 275 cm. of small bowel, has prompted me to submit the following case, which, tho not so extensive, is felt worthy of recording. This would be the 66th report of successful surgical treatment of mesenteric vascular occlusion. For a review of the literature, the reader is referred to the article by E. S. Koon, Jr., M.D. "Mesenteric Vascular Occlusion", J.A.M.A., Vol. 143, Number 16, August 1950, page 1408.

REPORT OF CASE

A 53 year old white female was admitted to the Virginia Beach Hospital at 6:45 P.M., 6-15-50, complaining of agonizing pain in the midepigastrium. She was well until 2½ hours prior to admission, at which time she drank a glass of milk, and immediately experienced a sudden sharp pain in the rectum. The pain gradually worked up to the midepigastrium, and in about 15 minutes became so severe that she was prostrated. The pain radiated around both sides to the back. She felt nauseated, but did not vomit. A physician was called right away, and he gave her morphine, grains ¼, without relief. This was repeated in a few minutes, but the pain continued and she was brought to the hospital. She vomited once enroute. She had had a normal bowel movement that morning. There were no urinary symptoms; no chills or fever. She had had upset stomach intermittently for the past six weeks, which was always relieved by milk of magnesia. Past history revealed a total hysterectomy, bilateral salpingectomy, and appendectomy in January, 1949; X-ray treatment for goitre 35 years ago: otherwise negative.

Upon admission the patient appeared acutely ill, although the symptoms seemed out of proportion to the physical findings. The temperature was subnormal (97), pulse 80, respiration 20. Blood pressure was 100/70, heart and lungs normal. The abdomen was acutely tender in the midepigastrium. There was no spasm, no distention, and no masses. The lower midline scar was intact. Rectal examination revealed no tenderness, masses or spasm. A tentative diagnosis of acute pancreatitis was made, and gastric suction was instituted. With heavy sedation she spent a fair night. In the morning she appeared worse. T. P. R. 99.2: 110:28. The abdomen was more tender, and the pain apparently more severe in spite of morphine. There was no spasm or rigidity, and very little distention.

The blood amylase was normal. An upright film of the abdomen revealed no free gas under the diaphragm, and no evidence of intestinal obstruction. Wbc. 14,000 with 87% polys. The hemoglobin was 97%, and the Rbc. 5,210,000.

In spite of the negative X-ray, an intestinal obstruction due to the previous operation was suspected, and the surgical consultant was asked to see the patient. He agreed with the diagnosis, and recommended immediate exploration.

With the patient under spinal anesthesia, the scar of the previous incision was excised, and the peritoneal cavity explored. Lying free in the abdomen in an area entirely devoid of adhesions was an 18 inch loop of small bowel, near the junction of the jejunum and ileum which appeared devitalized. The proximal portion was more severely involved than the distal, the first inch being practically gangrenous. The small bowel was nowhere distended, and the involved segment was entirely collapsed, the proximal portion presenting a sharp line of demarcation; the distal portion had only spotty involvement. Arterial pulsation in the mesentery was good. There was a diffuse capillary thrombosis in the mesentery near the bowel extending for about 6 inches, the first inch being practically gangrenous., There were two small band adhesions deep in the pelvis between the bladder peritoneum and the terminal ileum, and two between the colon and bladder peritoneum, but there was no obstruction.

Decision was made to resect a portion of the small bowel approximately 18 inches long. A side to side

anastomosis was performed and the abdomen was closed in layers with chromic catgut and heavy cotton tension sutures.

Pathological examination showed only hemorrhagic infarction with beginning necrosis.

Post-operatively the patient was started on gastric suction, penicillin and streptomycin q. 3 h., sedation and intravenous fluids. The post-operative course was uneventful, temperature never rising above 100. Suction was discontinued on the 5th day, and oral fluids were allowed. Sutures were removed on the 6th day, and penicillin and streptomycin were discontinued on the 7th day.

She was discharged on the 15th post-operative day, at which time she was taking a full diet, and her only complaint was of weakness. She has been followed closely in the Out-Patient Department, and has had no symptoms referable to the operation.

U. S. Moves Wounded by Air Quickest, Safest Way.

Wounded G. I.'s in Korea are being returned to the United States in the quickest and safest way possible—by air, an article in the current May issue of *Today's Health* pointed out.

Air evacuation, an emergency feat prior to World War II, has become "standard operating procedure," according to Dr. Howard A. Rusk and Eugene J. Taylor of New York City, authors of the article. They estimate that 97 per cent of the wounded have been moved to the United States in this manner.

"Important first as a lifesaving measure, the speed of air transportation of the sick and disabled has many other advantages—comfort, shorter hospitalization, great economy and the saving in man-hours of medical personnel," they said.

A recent study of the Joint Chiefs of Staff was cited which estimated that more than 20 times as much medical personnel is required for moving patients by hospital ship as by air. As an example, a ship that moves 300 patients a distance of 10,000 nautical miles requires 126 medical personnel, including 8 doctors. Three hundred patients transported the same distance by air require but three to four medical personnel and no doctor at all. This means 282 man-days by air, in contrast to 5,670 man-days by hospital ship. The article continued:

"In addition to speed, trips by air have the great advantage of flexibility and frequency. C-54 evacuation planes travel approximately ten times as fast as a hospital ship, require only 34 patients instead of 300 to make up a full load, and may travel in any direction over land or water. Greater trip frequency eliminates need for large holding hospitals overseas and long delays in getting patients back to the United States."

How helicopters and small liaison planes make it possible to pick up the wounded within a few feet of where they fell and move them back to the various airstrips, also was described.

"Although the helicopter is particularly adept at this, its usefulness is limited by its short range of flight. To overcome this obstacle, the Air Force has been experimenting with hooking on to helicopters in mid-air and towing them with long-range planes. Arriving above the wounded to be evacuated, the helicopter cuts loose from its tow, lands, picks up the wounded, rises again and is hooked on to the long-range planes for a tow-back to the airstrip."

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CLINICOPATHOLOGICAL REPORTS

From the Case Records of the Medical College of Virginia and the University of Virginia Hospitals.

HARRY WALKER, M.D., *Editor*
WILLIAM KAY, M.D., *Associate Editor*

CASE NO.: B-98342

A three months old, white female child was referred to the hospital because of failure to gain weight, and because of gradually increasing abdominal distention. The baby was born in a local hospital, and apparently was perfectly normal at birth. However, at the age of three weeks the child was given to a foster mother and she described the child, at this time, as being "nothing but skin and bone", and weighing less than 6 pounds. The foster mother was unable to give any other history of the patient prior to the time of her adoption. At this time a formula consisting of evaporated milk, Karo and water was given the child, but due to the onset of diarrhea, Dextrimaltose #3 was substituted for the Karo. (Formula—evaporated milk ounces 8, water ounces 18, Dextrimaltose 5 tablespoons.) The infant apparently took this formula well, but gained weight very slowly. The foster mother noted that there was a gradual but persistent enlargement of the abdomen. There was no history of vomiting. The stools were described as being normal in number, volume, consistency, and color.

Physical Examination: Temperature 98. Pulse 148. Respirations 38. Length 51½ cms. Head 37 cms. Chest 32 cms. Abdomen at the umbilicus 37½ cms. Weight 8 pounds, 5 ounces. The child was fairly well developed, but poorly nourished and pale. The head appeared large in comparison to the face and the rest of the body. The fontanelles were open, and the suture lines separated. The frontal bones appeared small, while the occiput appeared large. No gross abnormalities of the eyes, ears, nose and throat were noted. The lungs were clear. Respirations were shallow and rapid. Examination of the heart revealed no abnormalities other than the rate of 148 per min. The abdomen was markedly distended and tympanitic to percussion. The superficial abdominal veins were prominent. There were tinkling peristaltic sounds over the entire abdomen. No masses could be palpated. External genitalia were normal. The extremities were essentially normal except for an increase in length of both little fingers in comparison to the other fingers. The neurological examination was essentially negative. The skin was pale with a scanty amount of subcutaneous tissue and was moderately dry. There was a slight icteric tint to the sclerae.

Laboratory Data: Hemoglobin 11.2 grams. Red blood count 3,510,000. White blood count 10,150, Polys. 49%, Bas. 2%, Lymphs. 45% and Monos. 4%. Urine: amber, acid, specific gravity QNS, 2 plus albumin, 2 plus sugar, negative acetone, microscopic showed many bacteria. Flocculation negative. Blood sugar 164 mgm.%. NPN 31 mgm.%. Serum proteins total 6.44, albumin 3.92, globulin 2.52. Stool examination revealed neutral fat to be present, no starch present, and the specimen gave no reaction to the x-ray film test for trypsin. Flat and upright films of the abdomen showed gas in the large bowel to the upper descending colon and gas in the small bowel. The generalized hazy appearance of the abdomen on the upright films suggested a possibility of ascitic fluid. The spleen and liver appeared to be somewhat larger than normal with both domes of the diaphragm elevated. The lung fields were clear and on an AP film the heart was not unusual. Long bone films of both arms and legs showed no periosteal elevation or other pathologic change. Two days after admission abdominal paracentesis yielded 350 ccs. of straw-colored fluid, with a protein content of 850 mgm.% and a specific gravity of 1.008. Following paracentesis the liver was found to be palpable at approximately 2 to 3 fingers breadth below the coastal margin and was described as being very firm and leathery. The patient continued to drain some ascitic fluid from the site of the paracentesis for the duration of her illness. At this time a BSP test showed 22% dye retention in 80 minutes. Three days after admission a glucose tolerance test was reported as follows: Fasting 170, half hour 216, one hour 315, two hours 384, three hours 295 mgm.% respectively. Other laboratory studies were as follows: Cephalin flocculation 3 plus. Thymol turbidity 3. Serum

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bilirubin 4.3 mgm. direct with a 4.4 mgm. total.

The hospital course was essentially an afebrile one with the pulse varying between 95 and 120 and respiration from 25 to 40. The infant took its formula well while in the hospital, but the weight dropped following paracentesis to 6 lbs., 13 ounces, and continued to drop until on the day of death the child weighed 6 lbs., 2½ ounces. No vomiting or diarrhea was noticed, but the infant was listless and fretful at times. Five days after admission the child expired quietly.

DISCUSSION BY DR. CAROLYN MCCUE*.

We have under consideration today a very interesting little three-months-old white female. The social history in this case may not be quite so interesting as some others we have had discussed recently; in fact, I think if we had to surmise the story here we might be suspicious that we even had an illegitimate child. We know at least it was an unwanted child. We are also told that this child was born in a local hospital and that nothing was unusual about its delivery. We know it was given to a foster mother at three weeks of age and all our history is from her and this seems very meager. We know that the baby had had relatively poor care. We can assume from the protocol that the only food was this standard evaporated milk formula, with no vitamins, no cereals, no orange juice in the three-month period. This is certainly not the usual pediatric procedure on a three-month old child, particularly for one who has not been gaining or doing very well. But, I can not believe we can assume that the liver disease here is due to any alcoholic intolerance or any other social problem of this particular age.

The medical history tells us several very important things. In the first place, the child had a story of failing to gain weight; it weighed around 6 pounds at birth. At the time it came into the hospital it weighed around 8 pounds. After the paracentesis was done it went back to about 6 pounds; so far for all practical purposes it never gained anything over its birth weight.

It had a large abdomen which was one of the major complaints that was offered. Its large abdomen had been noted from the first when the foster parent received the child, and had grown progressively worse. The story we have is that the stools were normal in number, consistency and color, which covers a wide scope particularly in view of the facts that we get later. We hope that is a true statement and we are bound to presume that that fact is as stated. We are also interested in the fact that there are no neurological symptoms. This child had no spastic paralyses, no respiratory symptoms, and we hope all of that is as negative as it is stated.

On examination the child was afebrile, temperature 98°F., pulse 148 per min.—it is not very much out of line because it was three months old—the child was small and emaciated and with scanty subcutaneous tissue. As to her head, I think the observer would have you think that it was almost a hydrocephalic type shape of head with the sutures still not united, the fontanelles being large but there is no tension and the measurements themselves are certainly not grossly out of proportion for a child of that age.

It is important that the lungs are negative as I mentioned, both in history and on examination. The abdomen is the main site of our physical findings. There, the superficial abdominal veins are prominent, there is marked distention, which first we are not sure what the cause is, and then it is realized that it is probably ascites. We are told later in the protocol that, when 350 cc. of the transudate was removed, the liver was two or three fingers down, that it was firm and leathery in consistency.

It does not mention feeling the spleen. We will see what the x-ray says about that. Certainly it is questionable that the spleen is not enlarged too. We know there was a slight icteric tint, and the statement is made that the little fingers were slightly long in proportion to the rest of the hand. If the fundi were seen, no cherry red spots were visualized.

As to the laboratory studies: The blood count is certainly normal, 11.2 grams is average for a three month old child, 10.5 being the normal standard at this age. The rest of the findings are not out of line. The urine is notable for 2+ albumin and 2+ sugar, without acetone or diacetic acid and without bile and without cells. This is a rather odd combination of circumstances in view of facts that come along later.

We have stools which show neutral fat present. Artificially fed infants normally will have about 35% as neutral fat. Breast babies will have even more fat but this stool is not a greasy stool of undigested fat or a very offensive stool of undigested

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protein which you would be suspicious of in a case of pancreatic deficiency. We are told there is no starch in the stool, and normally some is found.

There is a negative x-ray film test for trypsin and I do not know whether everyone is familiar with that. A test was recently described by Harry Schwachman and his associates in August 1949, where the stool is diluted with a little sodium bicarbonate, incubated, and if you have trypsin present it is a very simple test to show the digestion by the enzyme on X-ray film. The trypsin in the stool will digest the gelatin of the x-ray film very promptly if it is incubated and you can see the enzyme activity. It is not 100% accurate and certainly on one examination I would not get too excited over the fact that the child did not show any trypsin in the stool, particularly in view of the fact that all the stools consistency and color were normal.

The Wassermann or the serological test for syphilis being negative in this case is very accurate because a flocculin test in a three-months old child should be reliable. While syphilis could conceivably explain all of these symptoms on a congenital syphilitic basis, I cannot feel we are justified in thinking very seriously of that in view of this negative test.

Blood chemistries show we have a normal NPN, serum proteins which are very slightly low, 6.58 being normal at birth, here it is 6.44. We have a little bit of lowering of the albumin and raising of the globulin but a very minimal amount with an AG ratio, 1.55 here, average being 1.7 at this age, so I do not think that is very marked.

I am very much impressed by this high blood sugar, 164 mgm.%, 170 mgm.% later, which we will talk more about later. Liver function tests also showed a BSP with 22% dye still retained at the end of an hour and a half. This is marked retention which should be all clear at the end of this time. Cephalin flocculation being 3.+ is another index of an unusual protein formation here; and thymol turbidity being another test of the same general nature, showing these globulins and proteins as we find in hepato-cellular jaundice. Thymol turbidity 3, where an infant's maximum is 0.77 normally. The serum bilirubin is 4.3 direct, or almost all direct, the type we would expect with an obstructive type jaundice, the type which has been separated from the protein element. The glucose tolerance test certainly is an extremely interesting thing in a baby and a very rare thing to find in a child of this age. Of course, we are not told whether this was all fermentable sugar or whether it was not fermentable sugar, and I understand this could all have been glucose or galactose or some other sugar, or it could be any combination of these things, but certainly it is a high fasting level, and the slow fall is in perfect keeping with the diabetic type that we are used to thinking of. Maybe Dr. Mandeville would like to show us the x-rays.

DR. MANDEVILLE*.

This is a three months old child. The long bones show no periosteal elevation. It is true you could have rickets or scurvy in a child below the age of six months but very rarely so. Therefore, we would suspect nothing in the bones. In only one case out of five do we find periosteal elevation in syphilis; therefore, I am afraid from the standpoint of the skeleton I can give no help.

Now, from a standpoint of the lungs: They were perfectly clear; the heart was not unusual from an x-ray standpoint. The abdomen appears to be very distended. The domes of the diaphragm are rather high, but neither one nor the other is higher than would be expected in a general abdominal enlargement. There is a suggestion of an enlargement of the liver. The spleen might be very slightly enlarged but not greatly so. I would not think the patient had intestinal obstruction. The spine is not markedly curved in any way. The sutures are not wide, I do not see any evidence of hydrocephalus.

There seems to be a slightly enlarged liver, possibly a very slightly enlarged spleen, and ascites. Otherwise the x-ray examination is essentially negative.

DR. MCCUE RESUMES:

I think it is rather interesting too, that in the prematures, where there is rapid growth, you might get rickets at this age. Since rickets would be in proportion to the rapidity of the growth, and with this child not growing you would not expect it.

As far as the hospital course is concerned, we are told that the child had a paracentesis and had a blood glucose tolerance test, done two days after the baby came in, and that she died on the 5th day. No mention of any therapy is made.

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In the differential diagnosis in this case, I was impressed by four important points: In the first place, I do not think you can get around the fact that this child had ascites to a very striking degree. The second thing is the baby obviously had some liver damage as is very evident from the total picture and the chemical tests we have in addition. I was very much impressed by the diabetic-like blood sugar curve, and the high fasting blood sugar, which I think probably is the most helpful clue that we have in this whole case—certainly a most unusual thing in a three months old child. The fourth thing is that I think we should mention the absence of trypsin in the stool. As I have said, I do not believe this is a case of cystic fibrosis of the pancreas although the child had wasting and a big abdomen. In view of the ascites and the obvious liver damage, too, the normal stool and the absence of the respiratory systems, I feel that we can pretty safely dismiss the possibility of a cystic fibrosis of the pancreas and look for something else.

The ascities obviously brings up the possibility of many different causes. I cannot conceive that this child had a cardiac origin, with the normal heart we see on x-ray and no mention of cardiac findings, no other signs of failure in the lungs, etc., or pericardial signs. I do not think we have to seriously consider the infectious origin of ascities, although I suppose we might at least think briefly of tuberculosis, which conceivably could give you ascites at this age. A tuberculin test was made but I do not believe tuberculosis would explain the rest of the picture here very well. Nutritional ascites would certainly give you far lower serum proteins and edema elsewhere other than just ascites.

While there is some albumin in the urine, it is not massive and there were no red cells and the other changes we really would expect in nephritis are absent.

Malignancies would be exceedingly rare at this age and would not, as far as I can see, explain the picture.

As far as the liver damage is concerned, the portal obstruction would certainly seem to be the most likely cause of the ascities and I think if you just look at this case from the beginning, you would certainly think of some sort of cirrhosis, particularly if you were not too concerned about the blood sugar pattern. We have several congenital types of cirrhosis, things like Wilson's disease or hepatolenticular degeneration, but that comes on with marked neurological symptoms in the teens as a rule. Hereditary angiomatosis has been reported to cause cirrhosis but that does not give you all the damage that we have here. The obstructive causes of cirrhosis, things like congenital atresias, choleocystitis, etc., would certainly give you clay colored stools with a very high icteric level and would give you the evidence of much more marked obstructive type of jaundice. Again this would not explain the blood sugar problem. We might mention the fact that it has been reported that inspissation of bile in the intrahepatic ducts in erythroblastosis was cystic fibrosis of the pancreas. As to congestive type of cirrhosis—we mentioned the cardiac failure and thrombophlebitis of the inferior vena cava—again we have no evidence to think very seriously about those.

Of course, we do not know if this child had any poisons—we do not presume it was given any arsenic, silicon, etc. or mushrooms or various other types that might have given it cirrhosis at this age. As you know, such things have been produced, mainly in animals, but we have no history to suggest that here. I do not suppose the child had any alcohol, and I do not know of any report where alcohol in the parent has given cirrhosis in the children, at this stage.

Infections, of course, are frequently associated with obstructive type of cirrhosis, or can occur alone leading to cirrhosis; notable we speak of syphilis which I feel is pretty well excluded in our seriological test.

Other types of cirrhosis, of course, are the extreme fatty infiltrations which are not a very rare thing. They usually do not give you the other associated symptoms which we have here.

I think we might mention here briefly infectious hepatitis, which has just recently been reported in infancy. Ascites is not the common finding. The stools are clay colored—there is more biliary obstruction and as a rule fever of an infectious disease, and not the blood sugar changes.

The reticuloendothelial diseases seem to me to probably be a little more logical trend of thought. The first part of this week I was all ready to discuss Acute Infantile Gaucher's Disease, which is a very rare sort of problem but which has certain features which could fit in with this unusual picture. It has

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been reported to show jaundice and diabetes and nephritis, all associated in an infant that appeared normal at first, then developed neurological signs; they have spastic paralyses and as a rule get a lot of laryngo-spasm and trismus. The only one I have ever seen had the striking finding of having great difficulty in eating due to laryngo-spasm and trismus, and I could not conceive that we could quite ignore the fact that this child had no neurological symptoms at all. I say again, this is a very rare disease and is a very different type of disease from the chronic Gaucher's usually thought of in older people; but of course both are due to this storage of kerasin which is one of the types of storage disease which we can get. The other types are Niemann-Pick disease and Hand-Schüller-Christian disease, of the storage diseases, but they do not fit in here nearly as well in an infant. If it had been an adult, someone would have thought of Hemochromatosis or bronze diabetes. It was one of the first things I looked up. Dr. Priscilla White reported it was never seen in children less than two years of age. That is very logical because it would take years for enough iron to be stored in the liver and spleen, etc., to accumulate enough to give you the physical picture we have where diabetes and cirrhosis are associated.

I was very much interested in looking up the differential diagnosis of glycosuria in children and found that 66% of all glycosuria in children was non-diabetic in nature. This brings me to my final suggestion: This is galactose intolerance, galactose diabetes or galactemia. Without the ketosis you are bound to think seriously of the type. Glycosuria can be caused by excessive carbohydrates in the diet. It can be caused by low renal threshold. It can be caused by von Gierke's disease. This disease should have been mentioned briefly under the reticuloendothelial system as another one of those rare sort of storage diseases where you get glycogen storage leading to large livers, spleens, often with enlargement of the heart at the same time. These children usually, though, in spite of these tremendous livers are very well developed, well nourished childen who get along remarkably well for years with the tremendous amount of glycogen in storage in the liver, in the heart and in the various other organs, with remarkably little laboratory evidence of any derangement of their liver function except for the low blood sugar.

It seems to me that this problem of the galactose intolerance might be a very reasonable one here, as on studying the problem further I could find nine cases, so far, where a picture has been described which compares well with our case today. The first one was described in 1917 by a German named Goppert who had three children in one family; two of them died less than two months of age with ascites, jaundice, tremendous abdomen, etc. The child he described was the one about two years of age, which he found with galactose, a non-fermentable sugar in the urine. The child had ascites, a large liver, wasting and mental retardation. After he found the galactose in the urine he took the milk away from the child and immediately be began to thrive. He first reported it and other people have noted the facts and there have been some other reports, a number of other reports since that time, each one pointing out the same general clinical features. They are Mason and Turner in 1933, Norman and Tashua in 1943, Mellinkoff in 1945, and Goldbloom in 1946.

It is interesting to notice that the galactose seems to be very poorly utilized or metabolized by the liver. Exactly what happens to it or whether it is stored in the liver or whether it has a toxic influence on the liver, I have been unable to determine, but each one of these report positive cephalin flocculation tests, ascites, very large livers and, interestingly enough, in each one of them the 2+ albumin and the 2+ sugar in the urine is very characteristic. Of course, as soon as the galactose is removed from the diet, that immediately disappears. Mental retardation and cataracts often occur too.

These writers have theorized different underlying causes. To me the most likely one is Mellinkoff; that galactose probably has a toxic effect on the liver cells and for that reason acts in a more damaging way than glycogen storage does in causing a picture very much like a portal cirrhosis, with obstruction and ascites and later parenchymal liver damage with the changes in the liver function tests such as we have described here.

As far as the kidneys are concerned, the best thing seems to be that there is probably an altered protein that is excreted which shows up as 2+ albumin on the routine tests and which, as soon as the galactose is removed, disappears immediately.

As far as I have been able to find, all these chil-

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dren that have been reported have lived and have been followed on galactose-free diets on which they would thrive and improve after being extremely emaciated and hungry and having the physical picture described here before. I do not think one of the clinicians was intending to give me a clue but he was very helpful when he told me he thought this child was going to live. Once milk was removed from the diet, if the child had survived a few more days, it should have had a good prognosis.

I would like to suggest that I think this child had galactemia, with liver damage, portal obstruction, ascites, and probably cirrhosis on a toxic basis. I will be very interested to know whether there is galactose stored in the liver, or just what is the pathological finding. I believe she died with liver failure and emaciation.

Fig. 1. Enlarged spleen with prominent connective tissue markings.

HOSPITAL DIAGNOSIS: Galactemia.

DR. MCCUE'S DIAGNOSIS: Galactemia.

PATHOLOGICAL REPORT: Dr . Gordon R. Henningar*.

At necropsy, the infant was poorly nourished and developed. On opening the peritoneal cavity, approximately 50-75 ccs. of clear, serous fluid was evident. The liver weighed 180 gms. (normal—130). The shape was not disturbed and the surface finely nodular. A green color was imparted to the organ. On cut section, the edge was irregular due to small humps, separated by strands of connective tissue, causing the capsule to be retracted from the surface. The hard consistency of the liver was easily appreciated. A careful dissection revealed no abnormalities or occlusion of the gall bladder and bile ducts. Chemical analysis on a representative portion of the

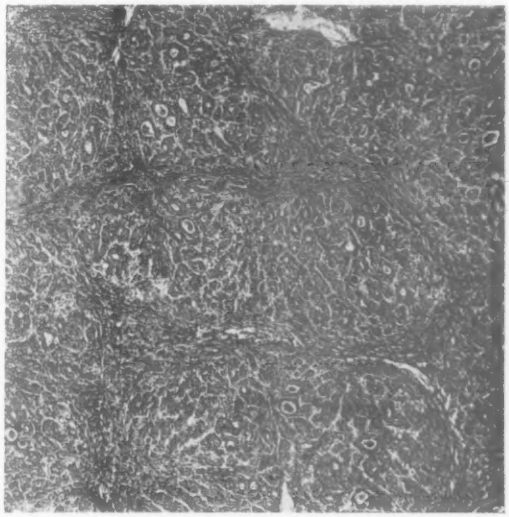

Fig. 2. Small nodules separated by connective tissue.

liver revealed an increased amount of lipid and diminished content of glycogen. A detailed account on this analysis and the pathological findings are to be published. The spleen was enlarged weighing 90 gms. (normal 40-50 gms.). On cut section, the

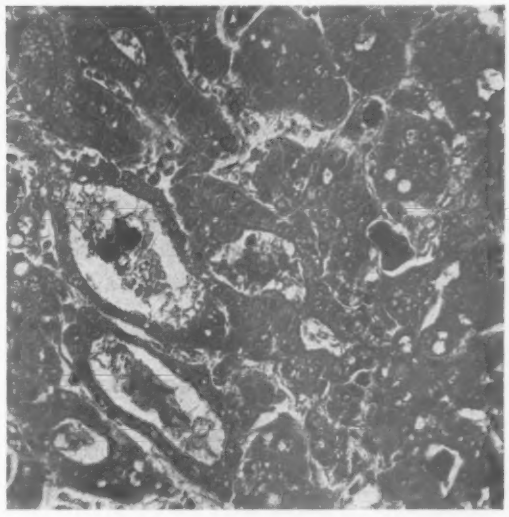

Fig. 3. Peculiar acinar structures containing bile thrombi. Note also vacuolation of liver cell cytoplasm (fat droplets).

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red pulp was red and prominent. The Malphigian corpuscles were small and widely separated (fig. 1). The trabecular markings were very distinct. One had the impression that the connective tissue content of the organ was increased. The remainder of the organs were not remarkable. Some twenty-five sections of the brain were cut after formalin fixation. In none of these did we see any lesions. We were particularly eager to assoicate this case with the disease known as hepato-lenticular degeneration of Wilson.

Microscopically, the liver presented a most challenging appearance. The sections showed the organ to be composed of small nodules separated by thin strands of connective tissue (fig. 2). Within the nodules one saw many acinar structures, containing bile casts or bile "thrombi" (figs. 2 and 3). These may be just markedly distended bile canaliculi, obstructed by inspissated bile. In the portal areas many proliferating and proliferated bile ductules were observed (fig. 4). In other areas large sud-

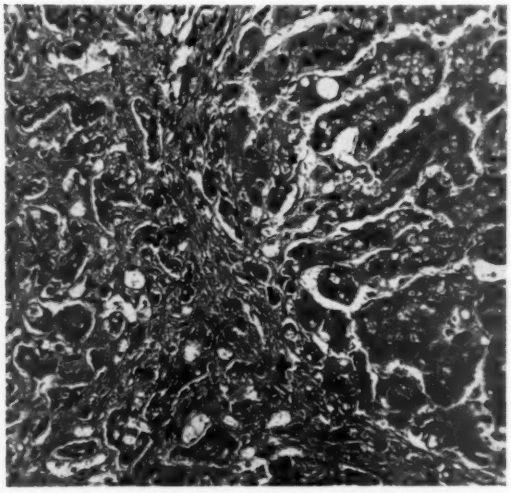

Fig. 4. Proliferated and hyperplastic bile ducts in portal spaces.

anophilic droplets were noted markedly displacing liver cells (fig. 5). Periodic-acid-Schiff positive granules were seen in a few cells. Sections were fixed in formalin, Bouin's, Zenker's, formol-alcohol, absolute alcohol. Special stains included Best's carmine, P.A.S., Masson trichrome, Azocarmine, P.T.A. The nature of the liver lesion, as well as its classification defy clarity. In some respects, it resembles Laennec's cirrhosis; in other respects, biliary cirrhosis. The possibility of it being a congenital malformation (peculiar congenital cirrhosis) must be entertained. The criteria for making the

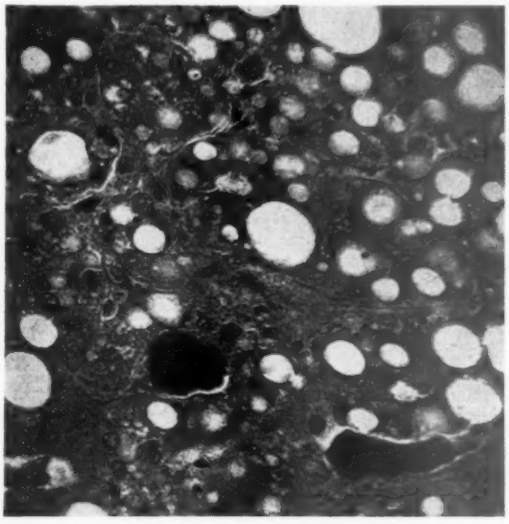

Fig. 5. Large sudanophilic droplets in the liver cells. Two large bile thrombi are present.

histological diagnosis of congenital post infectious hepatitis have not been elaborated as yet (if such occur).

The spleen, microscopically, showed thick walled splenic sinuses and could be interpreted as the spleen of early portal and splenic vein hypertension (Banti's spleen). No Gamma-Gandy (siderofibrotic nodules) bodies were seen.

The nature of galactemia with liver cirrhosis is not understood. The theories comprise:

(1) Galactose is a toxin causing hepatic necrosis followed by cirrhosis.

(2) A primary disturbance in galactose metabolism causing interference with carbohydrate and fat enzymatic systems leading to the accumulation of fat in the liver, followed by cirrhosis.

(3) A congenital and familial cirrhosis with the galactemia resulting from inability of the diseased liver to metabolize the galactose.

There have been four autopsies to date on patients with this disorder. Microscopically, our case resambles these. We believe, as a hypothesis, that there may be a disturbance in the content of the bile so that it becomes more viscous and plugs up the bile

ductules, leading to obstruction and biliary cirrhosis of intrahepatic origin. We feel that the galactemia is secondary to the liver damage and not the primary cause of the liver damage. We gave parenterally massive doses of galactose to rabbits over a period of fourteen days, then they were sacrificed. Histological examination of the liver revealed it to be literally "stuffed with glycogen" and no areas of necrosis were seen. Furthermore, we now have one infant of four months of age with congenital absence of the common duct and biliary cirrhosis (observed at operation) who is running a galactosuria.

Since the first description of this entity by von Reuss[1] in 1908, studies of the liver histologically, have been made by Bell[2] and Donnell[3].

PATHOLOGICAL DIAGNOSIS: Galactemia with cirrhosis of liver, splenomegaly, ascites, physical retardation, lipid pneumonia, fatty infiltration of heart.

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3. Donnell, G. N., Lann, S. H.: Galactosemia. *Pediatrics* **7**:503, 1951.

Reports Better Results from Intravenous Use of ACTH.

Intravenous injections of ACTH in greatly reduced doses produce equally as good or even better results than larger doses of the drug injected intramuscularly, according to a study made at the Veterans Administration Hospital, Long Beach, Cal.

A joint report on the intravenous use of the hormone was made in June 9 J. A. M. A. by Drs. William Mandel, Michael J. Singer, H. Rudolph Gudmundson, Lester Meister and Frederick W. S. Modern.

The doses used were only one-tenth to one-twentieth of those previously used, the doctors said. Commenting on the results of the treatment of 25 patients, all are responding "adequately," they said:

"No patients were refractory to the hormone when given by this method. There were no patients who relapsed while being treated with adequate doses. There were no patients who were refractory on subsequent treatment."

Subsequently, they treated 20 additional patients and the earlier results were confirmed.

The patients were treated for periods of from two to 42 days for a variety of conditions, including rheumatic arthritis and bronchial asthma.

"Ten patients had previously received the drug in intermittent intramuscular injections," the report said. "In these the response to intravenous infusion of the hormone was usually more prompt and complete. Remissions after cessation of intravenous treatment were generally more prolonged."

The obvious advantage of the intravenous form of administration was its relative economy, they pointed out. However, patients must be followed closely when this method of administration is used, for undesirable side effects may occur more frequently and with greater rapidity. The absence of refractoriness on the part of patients suggests that this condition may be in part related to the mode of administration, they said.

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PUBLIC HEALTH

L. J. ROPER, M.D.
State Health Commissioner of Virginia.

The Rabies Problem

According to the Bureau of Animal Industry of the United States Department of Agriculture, there were 7,910 cases of rabies reported in the United States in 1950. The distribution of these cases by species of animal is shown below.

REPORTED CASES OF RABIES IN UNITED STATES—1950

Dogs	Cattle	Cats	Swine	Sheep	Horses	Goats	Misc.*	Man	TOTAL
4,979	948	428	85	48	33	5	1,375	9	7,910

*Miscellaneous includes coyote, fox, rabbit, mice, gopher, ground squirrel, rat, squirrel, skunk, wild cat, raccoon, opossum, mule, ferret, lynx, ox, hamster, civet cat, mole, ground hog, deer. The fox was specified in 152 cases.

The distribution of reported cases in Virginia and adjoining states is shown below:

REPORTED CASES OF RABIES IN VIRGINIA AND ADJOINING STATES—1950

Virginia	North Carolina	Tennessee	Kentucky	West Virginia	Maryland	District of Columbia	Delaware
94	171	284	845	269	3	1	0

The present policy in Virginia in rabies control is to encourage the passage of local suppressive measures when rabies occurs.

The principal suppressive measures advocated during incidence of rabies are:

1. Destruction of all stray dogs.
2. Temporary quarantine of all dogs.
3. Immunization of all dogs.

Since, however, the mad dog or other rabid animal recognizes no political or geographic boundaries, it is becoming increasingly apparent that before rabies can be eliminated in the Nation there must be fairly uniform and efficiently enforced state regulations in all states.

Following dog bites the physician must decide between the relative danger of the development of rabies and the occurrence of serious reactions to vaccine administration.

It is agreed that rabies can be transmitted from rabid animals to man only by the direct inoculation of fresh saliva by means of a bite through the skin.

For guidance in treatment, a rabid animal is defined as one which:

1. Is proved rabid by laboratory examination.
2. Is clinically rabid.
3. Dies from unknown cause (without laboratory examination) within seven days after biting.
4. Disappears after biting—and rabies is present in community.

Even with these criteria in mind, it is not agreed that the administration of anti-rabic vaccine is 100% efficient.

Recently in Illinois, in a rural area with no recent history of rabies, a five year old white girl was bitten on the leg by a stray cat. Laboratory tests were impossible because of the decomposed condition of the head when received at the laboratory. Anti-rabic vaccine therapy was instituted nine days after the bite—clinical symptoms were observed fifteen days after the bite and the child died twenty-one days after the bite. This case demonstrates that rabies infection and death in the human can and does occur despite reasonably prompt administration of anti-rabic vaccine.

Furthermore, it has been demonstrated that there is always some possibility of severe post-vaccination reaction, paralysis and death.

We are brought to the conclusion that the prevention of rabies in man implies its elimination in animal reservoirs. The principal control steps are:

1. The elimination of stray animals.
2. Regular vaccination of household pets.

These measures have proved successful in eradicat-

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ing rabies in England and the Scandinavian Countries where the disease was once as common as it is in the United States today.

THE MORBIDITY REPORT OF THE BUREAU OF COMMUNICABLE DISEASE CONTROL

	May 1951	May 1950	Jan.-May 1951	Jan.-May 1950
Brucellosis	7	3	27	26
Diarrhea and Dysentery	101	213	852	1,387
Diphtheria	11	4	63	83
Measles	3,047	626	10,208	1,638
Meningitis (Epidemic)	15	9	70	70
Poliomyelitis	1	2	15	15
Rocky Mountain Spotted Fever	7	4	7	4
Scarlet Fever	64	55	578	475
Tularemia	2	0	20	18
Typhoid and Paratyphoid	1	4	19	28

MEDICAL SOCIETY OF VIRGINIA

Public Relations

Doctors Advise Selective Service

The Virginia Advisory Committee to Selective Service was created in the early fall of 1950. Originally appointed by the Governor, the committee is now composed of Dr. J. M. Emmett, Dr. Harvey B. Haag, Dr. Waverley R. Payne, Dr. L. J. Roper, and Dr. W. Henry Street. Dr. Emmett heads the committee of four physicians and the representative of the Dental profession, Dr. Street.

Some difficulty is met in an attempt to define the diverse functions of the Virginia Advisory Committee. However, its basic reason for existence might be agreed upon. In the vast governmental machinery now operating preparatory to national defense, a need naturally arose for professional medical personnel, especially in the armed forces. Coexistent with such military need was the equally important necessity of maintaining reasonable minimum health standards for the civilian population.

The National Advisory Committee to the Selective Service System evolved to effectuate the needed balance. The Virginia Advisory Committee to Selective Service is a component unit of the national set-up.

Functioning in various vantage points throughout the state, the Local Advisory Committees, consisting of nearby practicing physicians, do the bulk of personal study concerning individuals subject to the Selective Service Act. Such local committees advise Dr. Emmett's State Committee which in turn recommends to Selective Service that the particular physician be deferred or not be deferred on the basis of relative essentiality. It is well noted that the Virginia Advisory Committee has no power to defer persons but acts only in an advisory capacity.

On occasions the State Committee is requested to make essentiality recommendations to various branches of the armed forces. This need arises in the case of physicians commissioned by one of such branches or attached to the armed forces reserves.

Public Law 779 provides for the induction of physicians, dentists, veterinarians, and allied specialists as non-commissioned personnel. So long as volunteers for commissions in the Medical Corps meet the military demand, Public Law 779 will not be used, according to Selective Service officials. Second Army records show that Virginia ranks second in the entire military district in proportional number of medical personnel voluntarily accepting commissions. Volunteers receive an additional $100.00 monthly salary.

Since last fall the Committee has made approximately eighty recommendations for deferment of physicians, dentists and veterinarians. Such recommendations were limited to July 1, 1951 in every case. On May 17, 1951 the Advisory Committee met for the purpose of reviewing the eighty odd cases previously processed. Some forty were recommended for extended deferment to expire January 1, 1952.

At the request of the National Advisory Committee the Virginia Committee is presently engaged in processing reserve officers in the several armed forces components. A long range project, the study is devised to show the available reserve strength of all service branches.

Committee members have devoted extensive time and effort in this important phase of defense mobilization without remuneration. A real service is being rendered the civilian population of Virginia as well as the armed services.

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MENTAL HEALTH

JOSEPH E. BARRETT, M.D.,
Commissioner, Department of Mental Hygiene and Hospitals

The Family Approach to Mental Health*

It is common observation that, closely associated with the patient who has mental disorder, there is apt to be an individual with a rigid, domineering or even punishing personality. Good mental health is primarily a matter of sound interpersonal relationships established first as a child within the family group.

From the emotional security developing within his own psyche, a child reaches out to relate himself to others. As his inner security fails, his interpersonal relationships become unsatisfying, or are destructive to others, or sought solely within the realm of fantasy.

The family is the unit for concern in the problem of these interpersonal relationships, of mental well-being. And the family doctor, by name and tradition, is the first counselor in matters of family health.

Tradition paints the family doctor as a paternalistic gentleman with a goatee. And in our country's folk lore there have been three such benevolent gentlemen with whiskers: Santa Claus, Uncle Sam, and the family doctor. But the specialists have clipped the doctor's whiskers, and Santa Claus has never tried to enter the medical field.

To be a family doctor implies belief in the institution of the family, even the modern one which is pessimistically described as a divorce-ridden, contracept-devoted group of individuals bossed by its meanest member.

Human processes of being born, growing up, mating, and aging are fixed. And equally unchanging through our changing mores are the human emotions of love and possession, parental pride and protection, and grief and despair. These emotions must find their frustrated way to expression through living experience.

The problems they present are the materials confronting the family doctor as a cause or complication of the physical malady he is called upon to treat. It is within his professional judgment to use his own skills to heal the sick, or refer his patient to some specialist. This policy is equally applicable to the emotional factors complicating the disease: problems of personality disturbances, of marital discord, or of difficulties within the child parent relationships.

A mental health center is a combined family and health agency. Back in the days when the family doctor wore a goatee, he had one meek and docile daughter, the trained nurse. She did what she was told, and never talked back to her old man. Now he has a second daughter, the social worker. She is a flip young thing who defies all traditions and questions the unquestionables in life. But she is a smart girl who is educated and capable, and she is devoted to the same problems of interpersonal relationships as those complicating the medical ones her old man is treating.

In the past, the social work field has been a catch all for the untrained: a genteel means of livelihood for the socially prominent widow without funds, a play thing for the politically ambitious woman who is not capable of meeting the challenge of dynamic relationships within her own home, or a vicarious form of feeling for the sexually frustrated. The social work field to-day is led by trained men and women of integrity who have learned how to define the scope of their profession and work within the limitations of its skills and disciplines. Their contribution to treatment is primarily in a social work study of the personality interrelationships. As the patient is helped to adjust to reality problems in his life situation, the sounder elements of his personality are strengthened. He becomes a stronger, healthier person, more confident of his ability to direct his own life. The problem for the doctor and the social worker is one of accepting each other's point of view without damaging the professional standard of either one.

Mental health centers have a four fold approach. The first is the family doctor who instigates or seconds the referral. It is his job to help correlate the policy of the mental health center to the needs of his community. The examinations and treatments of the patients referred are in the hands of the special-

*Article prepared by Faith F. Gordon, M.D., Psychiatrist Director, Lynchburg Area Guidance Center.

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Medical Society of Virginia Cancer Committee

Chairman, GEORGE COOPER, JR., M. D.

Medical School Building, University, Virginia

Reprints of this and preceeding Bulletins may be obtained from this office

July, 1951

Case Reports

Case 1. A 35 year old woman, who had been aware of vague digestive complaints for some months, developed epigastric pain, nausea, and vomiting. She was hospitalized, and after five days of intravenous feeding, her symptoms were relieved. X-ray examinations of the heart and lungs revealed no abnormalities, but routine blood studies discovered a severe hypochromic anemia. She was treated for anemia, then discharged.

For the next year, her course was one of progressive weakness and fatigue. She suffered intermittently with mid-abdominal pain which was relieved by food. Then another episode of nausea and vomiting prostrated her. She spent seventeen days in a second hospital, and again was treated for anemia.

Two months later, a hard painful mass appeared in the left frontoparietal region. It grew rapidly to a diameter of four cm. She was then admitted to a third hospital, complaining of progressive weakness, mid-abdominal pain, and of the tender mass on her forehead. The previous finding of a severe hypochromic anemia was confirmed, and a gastrointestinal X-ray study, her first, demonstrated a carcinoma involving the stomach from cardia to pylorus. A biopsy of the painful mass on the forehead was reported as showing adenocarcinoma, probably of gastric origin. Palliative radiation relieved the pain in the metastasis but did not cause significant regression, and the abdominal distress was not dramatically improved. The outlook is, of course, hopeless.

Case 2. A 72 year old man was hospitalized for a prostatic resection. The hypertrophy proved benign, the operative result good. During his admission study, a story of recent vague upper abdominal fullness and distress was obtained. When the patient had recovered sufficiently from the prostatic resection, gallbladder and gastric X-ray studies were done. A polypoid tumor two cm. in diameter was demonstrated in the cardia of the stomach. The tumor and a collar of gastric wall was resected. The pathological report was leiomyosarcoma. Three years later, the patient is in excellent condition, with no evidence of recurrence of metastasis.

Comment: 1. As demonstrated by Case 2, even gastric malignancy can be discovered in a stage early enough to permit curative surgery. Defeatism on the part of the physician in his approach to the cancer problem insures only one thing — the defeat of that physician **and his patients** by cancer.

2. The discovery of early, curable cancer depends upon the physician's maintaining a high index of suspicion toward the disease and upon his prompt complete investigation of possible cancer symptomatology. The physician who does not keep cancer ever before him as a diagnostic possibility and who does not routinely use the physical examination and the X-ray, clinical and pathological laboratories, will not discover early cancer. He will be embarrassed and distressed when other physicians find advanced cancer in his referred patients.

3. In Case 1, a patient who was seen repeatedly by a number of physicians and who was actually hospitalized in two different institutions had cancer for well over a year before the diagnosis was made or even suspected. When finally recognized, her disease was hopelessly advanced. An X-ray examination of the stomach, indicated by the patient's symptomatology, would have revealed the real trouble at any time during that long period that she was under medical supervision.

4. Hypochromic anemia is a symptom, not a disease. Its discovery should have led to a careful search for a source of bleeding in the stomach or intestine. The possibility of cancer, as well as of such benign lesions as peptic ulcer, polyp, enteritis, etc., should have been considered.

5. The fact that the patient was a 35 year old woman was no excuse for overlooking a cancer of the stomach. This disease occurs frequently in women and not rarely in young people. Gastric lymphoma is more common in the young than in the elderly.

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ists at the center, and beyond his province to direct as they would be if he were consulting other specialties.

The three fold approach within the mental health center is headed by another physician, the psychiatrist. With the psychiatric social worker and the clinical psychologist, the team members work within the scope of their specialized professional training.

Emotional maladjustments resulting in problems of interpersonal relationships make up the major part of the treatment load of any mental health center. The problems are a family matter because of the family concern for the well being of one of its members, and because the interplay of feeling within the family group grooves the course of the symptoms. And a return to good mental health will require work on the part of the other members of the family as well as the patient and his therapist.

BOOK ANNOUNCEMENTS

Patterns of Disease. On A Basis of Physiologic Pathology. By FRANK L. APPERLY, M.A., M.D. (Oxford), D. Sc. (Melbourne), F.R.C.P. (London), Professor of Pathology, Medical College of Virginia, Richmond, Virginia. J. P. Lippincott Company, Philadelphia. 1951. xiii-456 pages. 50 figures and 37 charts. Price $8.00.

Cancer As I See It. By HENRY W. ABELMANN, M.D., Chicago, Ill. Philosophical Library, New York. 1951. 100 pages. Cloth. Price $2.75.

Handbook of Pediatric Medical Emergencies. By ADOLPH G. DeSANCTIS, M.D., Professor of Pediatrics and Chairman of the Department of Pediatrics, Post-Graduate Medical School, New York University-Bellevue Medical Center; Director of Pediatrics, University Hospital and Gouverneur Hospital, New York City. And CHARLES VARGA, M.D., Instructor in Pediatrics, Post-Graduate Medical School, New York University-Bellevue Medical Center; Assistant Attending Pediatrician, University Hospital and Gouverneur Hospital, New York City. St. Louis, The C. V. Mosby Company. 1951. 284 pages. With 51 illustrations. Cloth. Price $5.00.

Vitamin E. Annotated Bibliography 1940 to 1950. By PHILIP L. HARRIS and WILMA KUJAWSKI. The National Vitamin Foundation, Inc., New York, N. Y. 184 pages. Paper. Price $3.00.

Modern Foot Therapy. By R. H. GROSS, Pod.D., in collaboration with H. L. DU VRIES, M.D., S. L. ROBBINS, M.D., C. R. KRAUSZ, D.S.C., and L. E. WILSON, M.D. Edited by M. J. LEWI, M.D., pp. 720, illustrations 200. Price $9.50. New York; Modern Foot Therapy Publishers, 1948.

A different type of text has been written trying to encompass the whole field of foot therapy. The effort has been satisfactory as the volume is indeed a credit to modern chiropody. Its many contributors have in the thoroughness of their work met a need by bringing together within one binding all those various aspects of foot therapy for which it was necessary for the general practitioner to turn to individual volumes, many of which have not been adequately slanted for the specialty of the foot.

This book serves as the third edition of the "Practice of Podiatry", and each of its thirty sections has been written by an authority in that specific field. Physicians, as well as chiropodists, have contributed. The chapters, easily read, include differential diagnosis in detail; arthritis; peripheral vascular diseases; the newer antibiotics including penicillin, tyrothricin and streptomycin; gait analysis; verruca injection with a consideration of various technics and medications; minor surgery of the foot with instructions for post-operative care as well as the actual surgery; clinical photography with basic details; focal infection and dysbasia pododynia, a new and exciting concept of foot dynamics. The chapters on foot orthopedic and dermatological conditions are adequately covered for this broad field. This is the first text that stimulates the reader's interest in modern chiropodys approach to pediatric, geriatric and diabetic foot care. The inclusion of the latter is commendable because it was as a result of specialized diabetic foot care that chiropody has received the recognition of the medical profession. There are a good cross index, bibliographies of references and biographies of contributors.

"Modern Foot Therapy" is recommended not only as an excellent text for the student, but truly provides for the practitioner all that is new and valuable in treatment of diseases of the foot. It is indeed a book that the general practitioner can use for ready reference to obtain in a short time a concise and compact account of almost any clinical entity in modern chiropody.

A. P.

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EDITORIALS

Joseph L. Miller

IN BRINGING this series to a close, I am breaking a rule that I laid down for myself in the beginning. When some months ago I began this series of sketches in righteous indignation at the villainous attacks on doctors and their motives by Mr. Oscar Ewing in his propaganda for compulsory health insurance, I decided not to use as my subject anyone who was still alive. I had a feeling that it would be as embarrassing as, for instance, an old man telling a handsome woman to her face that she had wonderful eyes, or beautiful teeth or lovely hair. He could look at her with admiration without giving offense but to put that admiration into words might be quite another matter. So I hope Dr. Miller will forgive me for breaking my rule in his case.

Dr. Miller is such a beautiful example of a general practitioner who has led a busy, useful life in an-out-of-the-way place and at the same time earned for himself a nation wide reputation on account of his work on cultural medicine. He has made no discovery that has saved the lives of millions of people, as some of my medical worthies have, nor has he opened up new fields of medicine, but he has saved his hundreds, by his own skill and knowledge and he and his like have earned the gratitude of the public by their devotion to duty in places where it is generally thought the chances for fame and fortune are slim. But is this really so? Even in my small collection of medical heroes, Jenner was a country doctor, and Koch began his epoch-making discoveries as a general practitioner. Ephraim McDowell and John Peter Mettauer were country surgeons.

Like many of the subjects of these sketches, Dr. Miller is a Virginian, born October 10, 1875, in Macon County, West Virginia, of Virginia parents. He graduated in medicine from the University College of Medicine in 1900. While a student in Richmond he began his great collection of medical books and illustrations with the purchase of Dr. James McClurg's student's notes of Thomas Young's lectures on midwifery at Edinburgh, 1769-70. Dr. Young was professor of midwifery at Edinburgh and Dr. McClurg was the first president of the Medical Society of Virginia. A note on the fly leaf of the MS, in Dr. Miller's handwriting states that he paid 25 cents for the volume and that he had it bound in Morocco by Reese David.

Upon graduation, Dr. Miller took a contract practice with a coal mining company as the quickest way to make some money to pay for his medical education. He has been in Thomas, West Virginia, ever since. He has put Thomas on the map, at least in certain European circles. The story is told in Richmond that when his oldest son married, his employer, the Holland American Steamship Line, gave him a trip to Europe as a wedding present. On his way home, young Mr. Miller stopped off in London to get his father a present. He went to a rare book shop and picked out a volume for five pounds, and asked the dealer if he could mail it, as his luggage was all packed up. When he wrote on a card "Dr. Joseph L. Miller, Thomas, West Virginia," the dealer said "Your father has this book", and found a less expensive one that he thought his father would like. Dr. Miller, himself, told me a story on Dr. Friedenwald that illustrates the same point. It seems that Dr. Miller was in Baltimore to read a paper on some historic subject. After the meeting Dr. Friedenwald had a group around to his home. They were sitting in his rare book room and naturally talking about old medicine. Dr. Friedenwald showed an old book and remarked that he had been trying to find a portrait of the author and had a standing order with all the dealers in Europe.

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"I have one", said Dr. Miller, "and will be glad to send it to you when I get home." Dr. Friedenwald could not believe it until he actually got the picture. For years Italian dealers had been sending Dr. Miller a bale of pictures about once a year and Dr. Miller would take what he wanted and return the balance.

I once asked Dr. Miller if he had ever been to Europe and he replied that he had never had time to get away from Thomas, West Virginia. That is not literally true for I know that he has delivered lectures on old medicine at the Mayo Clinic as well as Omaha, Nebraska, Kansas City, Birmingham, Vanderbilt University, the Chirurgical Faculty of Maryland and the College of Physicians of Philadelphia. He was Founder's Day orator at the Medical College of Virginia in 1928.

Dr. Miller signs up his assistants for three years, for he says that is long enough. If the assistant is no good he wants to get rid of him in that time and, if he is good, he does not want the boy to be "stuck" in a mining town as he was. The people in Thomas, of course, do not share this view.

Thomas, West Virginia is a dusty coal mining town of two streets. When you drive along the upper streets you can look down the chimneys of the houses facing the lower street. One of my most cherished memories is a visit I paid him shortly after he gave the most valuable part of his library to the Richmond Academy of Medicine on condition that it would build a fire proof building in which to house the books. His gift was the cause of the Academy building a home for itself.

Dr. Miller is a widower and his children have started homes of their own in more congenial climes. The doctor lives alone in a flat above the drug store. A flight of outside steps leads to his door and to the Odd Fellows Hall on the third floor. He takes his meals at the hotel next door.

Mrs. Rucker and I drove up one day in the summer, arriving in the late afternoon. The windows of his flat were covered with coal dust blackened cotton sheeting, altogether a dismal setting, but we were in for the surprise of our lives. Something on the order of an Arabian nights transformation greeted us when Dr. Miller opened his door. The flat was furnished with beautiful old mahogany and good oil paintings and miniatures of the Miller family. Everywhere there were book cases bulging with rare books. After supper at the hotel, Dr. Miller began to show us his treasures. At first he brought out examples of fore edge painting and beautiful bindings. Mrs. Rucker, being tired from her long drive, retired early. Dr. Miller then showed his important medical items. It seemed to me that he still had an important library after having sent his best books to the Richmond Academy of Medicine. I remember his saying that he wished he could come back after he died and hear what was said at the auction of his books. He not only loves his books but he knows them. Many of them have a note pasted in the back to show the place the book occupied in the history of medicine or a short note about the author. Sometimes this is a clipping from the dealer's catalogues and sometimes it is a note in Dr. Miller's handwriting. In the *de Fabrica* of 1555, one of the books he gave the Academy, he has written a biographical note on every owner from Vesalius' time to the Richmond Academy of Medicine. This was not such a formidable task as it would appear at first glance for the owners of such a book were of necessity important personages with book plates which they had pasted inside the pigskin cover.

Thus we had a grand night and neither had any notion of going to bed until the wee small hours of the morning. Dr. Miller has developed a wonderful hobby for the evening of life. As he sits among his books, his spirit knows no boundaries of time or space.

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Jowett, in the introduction to the Dialogues of Plato, describes the philosopher as "the spectator of all time and all existence. He has the noblest gifts of nature, and makes the highest use of them. All his desires are absorbed in the love of wisdom, which is the love of truth. None of the graces of a beautiful soul are wanting in him; neither can he fear death, or think much of human life."

Hippocratic Oath

I swear by Apollo the physician, and Æsculapius, and Health, and All-heal, and all the gods and goddesses, that, according to my ability and judgment, I will keep this Oath and this stipulation—to reckon him who taught me this Art equally dear to me as my parents, to share my substance with him, and relieve his necessities if required; to look upon his offspring in the same footing as my own brothers, and to teach them this art, if they wish to learn it, without fee or stipulation; and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the Art to my own sons, and those of my teachers, and to disciples bound by a stipulation and oath according to the law of medicine, but to none others. I will follow that system of regimen which according to my ability and judgment, I consider for the benefit of my patients, and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to any one if asked, nor suggest any such counsel; and in like manner I will not give to a woman a pessary to produce abortion. With purity and with holiness I will pass my life and practice my Art. I will not cut persons laboring under the stone, but will leave this to be done by men who are practitioners of this work. Into whatever houses I enter, I will go into them for the benefit of the sick, and will abstain from every voluntary act of mischief and corruption; and, further, from the seduction of females or males, of freemen and slaves. Whatever, in connection with my professional practice or not in connection with it, I see or hear, in the life of men, which ought not to be spoken of abroad, I will not divulge, as reckoning that all such should be kept secret. While I continue to keep this Oath unviolated, may it be granted to me to enjoy life and the practice of the art, respected by all men, in all times! But should I trespass and violate this Oath, may the reverse be my lot!

MEDICAL CLASSICS, **3**:viii, 1938-39.

How many have read this medical classic? How many subscribed to it when they graduated in medicine? Formerly, taking the Oath of Hippocrates was as much a part of the graduation exercises in medicine as the defending a thesis was a part of the final examination. The public still thinks that every doctor subscribes to the Oath when he receives his M.D. degree. The only issue that has, so far, been tried by the Grievance Committee of the Medical Society of Virginia involved a reputed violation of the Oath. There was no question of fees or overcharging, for the complainant had not been a patient of the doctor who was being tried. She based her complaint upon the assertion that the doctor violated the Oath which he took when he graduated in medicine, in that he divulged a private conversation to her lawyer. None of the Committee knew about the "or not" in the phrase "Whatever, in connection with my professional practice *or not* in connection with it, I see or hear in the life of men, which ought not to be spoken of abroad, I will not divulge, as reckoning that all such should be kept secret." We thought that the Oath applied only to privileged communications in

the doctor-patient relationship. We were gratified that the public put so much store by the Oath.

As a matter of fact, while the Oath has been eliminated from the graduation exercises, every doctor subscribes to the Oath in principle when he joins the American Medical Association, for the Code of Ethics of the Association is based upon the Hippocratic Oath.

The World Medical Association has revised and modernized the Hippocratic Oath to cover such new developments as the Nazi medical crimes in the concentration camps in World War II. It is called the Geneva Oath, and must be subscribed to by each component society. It is further recommended that it be read by every doctor on receipt of his diploma to help impress upon him the "fundamental principles of medical ethics". It reads as follows: "Now being admitted to the profession of medicine, I solemnly pledge to consecrate my life to the service of humanity. I will give respect and gratitude to my deserving teachers. I will practice medicine with conscience and dignity. The health and life of my patient will be my first consideration. I will hold in confidence all that my patient confides in me.

"I will maintain the honor and the noble traditions of the medical profession. My colleagues will be my brothers. I will not permit consideration of race, religion, nationality, party politics or social standing to intervene between my duty and my patient. I will maintain the utmost respect for human life from the time of conception. Even under threat I will not use my knowledge contrary to the laws of humanity.

"These promises I make freely and upon my honor."

Dr. Henri Vignes

DOCTEUR Henri Vignes, Ancien Professeur a la Faculté de médecine de Paris, Accoucheur-Gynécologue Honoraire des Hopitaux, Professeur Honoraire a l'Ecole d'Anthropologie, Officier de la Légion d'Honneur, Croix de Guerre 1914-18, died at Poitiers March 15, 1951 in his 67th year. He was Co-Director of *Gynécologie et Obstétrique,* and a member of the editorial board of *Progrés médical, Journal des Praticiens, Semaine des hôpitaux, Sciences médicales, Revue de Pathologie comparée, Bulletin général de Thérapeutique, Revue de météorologie médicale, and Annales médico-chirurgicales.* He was also a regular contributor to *Presse médicale, Concours médical, and Revue sud-américaine de médicine et de chirurgie.*

Dr. Vignes was the author of a number of worth-while books on obstetrics and gynecology, notably "Physiologie obstétrical normale et pathologique" *Paris* 1923, "Physiologie gynécologique et médecine des femmes" *Paris* 1929., "Maladies des femmes encientes I Affections de tube digestif." (with G. Lauret & P. Olivier-Pallud) *Paris* 1935, "Maladies des femmes enceintes II Affections du foie, du pancréas maladies de la nutrition parois abdominales, péritoine" (with G. Lauret, Jean Olivier and P. Olivier-Pallud) *Paris* 1935, "Éclampsie et Éclampsisme" *Paris* 1948.

Since his graduation in medicine in 1914 with a thesis "Notes et recherches sur la menstruation", he has occupied a prominent place in French gynecology and obstetrics. We have for years been epistolary friends, ever since, in fact, the publication of our first article upon uterine contraction, when he wrote us a complimentary letter, and sent us a copy of his first book. He will be greatly missed in obstetrical circles.

XUM

Lonsdale J. Roper (1886-1951)

DR. L. J. ROPER, State Health Commissioner, died in the early morning hours of June 12th, of coronary thrombosis. He had an attack in January 1947, from which he made a good recovery. Except for some curtailment of certain physical activities he seemed as well as ever until June first, when he went to the hospital on account of a more serious attack.

Dr. Roper was born in New York, March 28, 1886. When he was six months old, his family moved to Norfolk County, and he has been identified with Virginia ever since. He attended William and Mary College and the University College of Medicine. He graduated in Medicine in 1907 and interned at the King's Daughters Hospital in Portsmouth. He engaged in private practice for a number of years and was coroner of Norfolk County during that time.

Dr. Roper was always civic minded and in Portsmouth he took great interest in the Kiwanis Club of which he was president at one time. In 1936 he was governor of the Capital District. He was honorary Life Member of the Portsmouth Club. He was largely instrumental in the founding of the Fresh Air Farm operated by the Portsmouth Kiwanians for underprivileged children. He was a thirty-second degree Mason, a member of the Scottish Rite and the Shriner's Khedive Temple in Norfolk. In World War I, he was a major in the Medical Corps.

In 1919, Dr. Roper began his career in public health with the establishment of Portsmouth's first city health department. In 1937 he became director of the venereal disease division of the State Health Department and the next year he was put in charge of the department's rural health division. In August 1946 the Governor made him Health Commissioner. He brought to that position a background of general practice. He had the firm conviction that in public health the cooperation of the practicing physicians was important. In his administration he adhered to that position and he had the whole hearted cooperation of the profession. That he had a remarkably successful administration, every one will agree.

He was a member of the American Medical Association, the Southern Medical Association, and the Richmond Academy of Medicine. He was an active fellow of the American Public Health Association and was president of the State and Provincial Health Administrators of North America.

In the Medical Society of Virginia he was a worker and, since the Health Commissioner is an ex-officio member of the Council, he had plenty of opportunity. He attended every meeting of that body and engaged in active discussions of many questions. He was a member of about one-half of the special committees of the Society. He will be greatly missed in its deliberations.

Dr. Roper is survived by his wife, Mrs. Florence Leslie Roper, a former superintendent of nurses at the King's Daughters Hospital in Portsmouth.

XUM

THE PRESIDENT'S MESSAGE

IN THE passing of our Executive Secretary and Public Relations Director, Mr. Henry S. Johnson, the Medical Society of Virginia has lost a valuable and loyal Executive. His place will be hard and very difficult to fill. I had begun to rely upon Mr. Johnson and his judgment and had looked forward to an easy and smooth-sailing administration. Instead a calamity has struck me.

I appointed Mr. Richard Nash, Mr. Johnson's assistant, as acting Executive Secretary until other arrangements can be made. To date, I have seven applications with numerous recommendations from throughout the state. I find the decision most too difficult for your President to make.

I have appointed a special committee to review the applications and interview the applicants and make their recommendations to the Council.

On this committee, I have named Dr. Vincent W. Archer as chairman. Dr. John T. T. Hundley, President-Elect, Dr. Guy W. Horsley of Richmond, and Dr. John M. Emmett, chairman of the Public Relations Committee.

A few months ago we appealed to the Councilors of each district to check on the number of physicians in their district to see how many, if any, are eligible to join the State Society. To date we have only heard from two districts. I appeal to you to make this check immediately. I personally feel that every qualified physician should be a member of his local and state medical society. He needs us and we need him. When a physician is trying to join any kind of organization outside of medicine, or to secure an important position, the first question they wish to know is "Are you a member of your local and state medical society?"

To chairmen of committees: Please call your committees together, lay your plans and have your report in the office preferably by July 15, but not later than August 1. Otherwise, they will not be printed in the Journal. The Special committees are a very vital part of any medical society, but they do not accomplish very much unless the reports are published and can be read and studied by the membership.

Councilors and Secretaries of local societies: Please check your membership to see if 1951 A.M.A. membership dues have been paid. We came near losing one of our delegates last year because we did not have sufficient paid members; unless they are paid up promptly, we run the chance of losing one yet.

To the members: Please check your reception room table to see if TODAY'S HEALTH is there. As this is one of the official publications of the American Medical Association, I feel that it is important and essential that physicians should support this journal. It brings a message to the layman that he would not get otherwise.

C. L. HARRELL, M.D.

XUM

SOCIETY PROCEEDINGS

The Warwick County Medical Society

Had a meeting on May 8, at the James River Country Club. There were 53 members and 2 guests present. Applications for membership were approved for Dr. Frank S. Beazlie, Jr., Dr. F. Ashton Carmines, and Dr. W. James Baggs.

Dr. W. W. Anderson, Jr., of Newport News, introduced the speaker, Dr. J. L. Calloway, Professor of Dermatology and Syphilology at Duke University, Durham, who spoke on "Contact and Therapeutic Dermatitis."

O. W. BOOTH,
Secretary-Treasurer

The Wise County Medical Society

Met May 9, at Hotel Norton.

The recent Kick Back of amounts paid for glasses and other EENT equipment was discussed. We could find no indication of this being any problem here.

Dr. N. J. Chew, Bristol, spoke on Experiences with Cortisone. Discussion followed. He thought ACTH and Cortisone about equal with ease of administration, especially since Cortisone can now be given orally, which makes Cortisone preferred in many cases. When either fails or proves undesirable he switches to the other.

Wyeth's latest cinema talkie—highlights of recent World Medical Conference in New York—held close attention for 35 minutes.

The Red Cross Blood Bank plan was approved and we shall co-operate.

Dr. F. B. Robinson, Oxford, Pa., and six visiting doctors from this area were guests. There was an attendance of twenty-nine. The President, Dr. George F. Sutherland, presided.

The next meeting will be on August 8th at Norton.

T. J. TUDOR,
Secretary

The Lynchburg Academy of Medicine

Held its regular monthly meeting on April 9th. This was a joint dinner meeting with the Lynchburg Pharmaceutical Society and was held at Oakwood Country Club. The guest speaker was Dr. Harvey B. Haag, Richmond, who spoke on "Relations Between Doctors and Druggists".

NEWS

Commencements of Medical Schools.

MEDICAL COLLEGE OF VIRGINIA.

The closing exercises of the College were opened Sunday evening, June 3, with Dr. Theodore F. Adams, of the First Baptist Church, preaching the baccalaureate sermon. The annual meeting of the Alumni Association was held on the 4th, with the social hour and banquet of the alumni that evening. A buffet luncheon for the Board of Visitors, faculty, graduates, parents, and friends was on Tuesday at the Social Center, with the commencement exercises that evening at the Mosque, followed by a reception and dance at the John Marshall Hotel.

Names of the graduates in medicine with hospital appointments follow:

MEDICAL COLLEGE OF VIRGINIA HOSPITAL, Richmond—Drs. Emma Jane Freeman, Adrian, W. Va.; Robert Finley Gayle, III, Richmond; Luther Johnston Hamlett, Ripplemead; Andrew Epes Harris, Jr., Blackstone; Francis Hunter McMullan, Richmond; Erma Jean Marra, Morgantown, W. Va.; Levi Old, Jr., London Bridge; Earl Elmore Roles, Jr., Lillybrook, W. Va.; Carl James Roncaglione, Amonate; Bernard Manuel Savage, Richmond; Victor Skorapa, Jr., Richmond; Julian Weinstein, Richmond; Yale Howard Zimberg, Richmond.

JOHNSTON-WILLIS HOSPITAL, Richmond—Drs. Lewis Bailey Hasty, Roanoke Rapids, N. C.; Edward Paul Jones, Mill Creek, W. Va.; Edward Arthur Lewis, Aylett; Henry Reece Little, Jr., Altavista; Arthur Joseph Martin, Richmond; Arthur Wainhouse Downing Mears, Belle Haven.

STUART CIRCLE HOSPITAL, Richmond—Drs. Aubrey Alphin Houser, Jr., Richmond; Homer Delmar Martin, Montrose, W. Va.; Norman Rock Tingle, Richmond.

LEWIS-GALE HOSPITAL, Roanoke—Drs. William Jasper Berry, Oak Hill, W. Va.; Ernest Jackson Kef-

fer, Jr., Roanoke; Joseph Frederick McGuire, Grundy.

ROANOKE MEMORIAL AND CRIPPLED CHILDREN'S HOSPITAL, Roanoke—Dr. John David French, Bluefield, W. Va.

NORFOLK GENERAL HOSPITAL, Norfolk—Drs. Walter Morris Brady, Lynchburg; Martel Jennings Dailey, Tye River; Norman Nash Hill, Jr., Norfolk; Walter Stanley Jennings, Fentress; Jesse Miller Tucker, Jr., Richmond.

DEPAUL HOSPITAL, Norfolk—Drs. William Francis Gibbs, Norfolk; Matthew Christopher Glynn, Jr., Portsmouth; Louis Henry Keffer, Jr., Portsmouth; Brooke Miller Moffett, Washington; Robert Macon Phillips, Colonial Heights; Philip Randolph Thomas, Richmond.

WINCHESTER MEMORIAL HOSPITAL, Winchester —Drs. Philip Winston Cowherd, Jr., Glade Spring; Oscar Bruton Darden, Jr., Richmond; William John Hotchkiss, Meyersdale, Pa.

RIVERSIDE COUNTY HOSPITAL, Arlington, Calif.—Dr. Hubert Elmore Batten, Portsmouth.

PRESYTERIAN HOSPITAL, Santurce, Puerto Rico—Dr. Carlos S. Berrocal, Mayaguez, Puerto Rico.

SAN JUAN CITY HOSPITAL, San Juan, Puerto Rico —Dr. Angelo Ismael Portela, Arecibo, Puerto Rico.

ST. MARY'S HOSPITAL, Huntington, W. Va.—Drs. Ralph Harold Boone, Wheeling, W. Va.; Ralph Burton Burner, Philippi, W. Va.; David Sheffer Clark, Huntington, W. Va.

CHARLESTON GENERAL HOSPITAL, Charleston, W. Va.—Drs. James David Brown, Craigsville, W. Va.; Preston Caldwell Davis, Beckley, W. Va.; John McClure Grubb, Charleston, W. Va.; William Dane Irvine, Lewisburg, W. Va.; Wilmer Grey Oliphant, Mount Hope, W. Va.; Paul Ernest Totten, Charleston, W. Va.; Robert Frederick Willis, East Bank, W. Va.; James Earl Wotring, Morgantown, W. Va.

ST. JOSEPH'S HOSPITAL, Phoenix, Arizona—Dr. John Fremont Bos, Buckhannon, W. Va.

TOURO INFIRMARY, New Orleans, La.—Dr. Courtney Cox Bowen, Tazewell.

CHARITY HOSPITAL, New Orleans, La.—Dr. Allan Campbell Oglesby, Richmond.

STATE OF WISCONSIN GENERAL HOSPITAL, Madison, Wis.—Drs. Miriam Willena Carmichael, Richmond; Charles Gordon Spivey, Jr., Columbia, S. C.

MICHAEL REESE HOSPITAL, Chicago—Dr. William Agee Cook, Jr., Madison Heights.

UNIVERSITY OF PITTSBURGH MEDICAL CENTER, Pittsburgh, Pa.—Dr. Merle Burton Davis, Fairmont, W. Va.

ST. FRANCIS HOSPITAL, Pittsburgh, Pa.—Drs. Elmer Francis McHugh, Morgantown, W. Va.; Albert Martin Valentine, Morgantown, W. Va.

ROBERT PACKER HOSPITAL, Sayre, Pa.—Dr. John L. Fairly, Jr., Richmond.

PHILADELPHIA GENERAL HOSPITAL, Philadelphia —Dr. William Calvin Grigsby, Jr., Bristol.

GOOD SAMARITAN HOSPITAL, Portland, Ore.—Dr. Julia Cecil Dickinson, Richmond.

JACKSON MEMORIAL HOSPITAL, Miami, Fla.—Drs. Calvin Hunter Frazier, Huntington, W. Va.; Frederick Ashley Gunion, Coral Gables, Fla., Abraham Lowell Newman, Phoebus; Paul Eugene Shutts, Vienna, W. Va.

DETROIT RECEIVING HOSPITAL, Detroit, Mich.—Dr. Richard Kelly Greenbank, Richmond.

HENRY FORD HOSPITAL, Detroit, Mich.—Dr. Clarence Edwin Hicks, Spring Hill, W. Va.

BROADLAWNS POLK COUNTY HOSPITAL, Des Moines, Iowa—Drs. Thomas Henry Jennings, Appomattox; Robert Frederick Schnabel, Huntington, W. Va.

JAMES WALKER MEMORIAL HOSPITAL, Wilmington, N. C.—Dr. Joseph J. Lindley, Snow Camp, N. C.

REX HOSPITAL, Raleigh, N. C.—Dr. Joe Watson Trent, Ft. Blackmore.

WORCESTER MEMORIAL HOSPITAL, Worcester, Mass.—Drs. Thomas Day McCahill, Portsmouth; Edward Woodward, Jr., Staunton.

CINCINNATI GENERAL HOSPITAL, Cincinnati—Dr. Harold Wilbur Miller, Jr., Woodstock.

FLUSHING HOSPITAL AND DISPENSARY, Flushing, N. Y.—Dr. Pamela Ross Moore, Lewisburg, W. Va.

ST. LOUIS CITY HOSPITAL, St. Louis—Dr. James Lee Northington, LaCrosse.

BETH ISRAEL HOSPITAL, Boston, Mass.—Dr. Albert Julian Wasserman, Richmond.

UNIVERSITY OF MINNESOTA HOSPITAL, Minneapolis—Dr. Harry Charles Walker, Jr., Richmond.

WILLIAM BEAUMONT GENERAL HOSPITAL, El Paso, Texas—Dr. Edward Henry Currie, Charlotte, N. C.

U. S. NAVAL HOSPITAL, San Diego, Calif.—Dr. Joseph Herbert Britton, Richmond.

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U. S. NAVAL HOSPITAL, Great Lakes, Ill.—Dr. Fred Ray Edens, Norton.

U. S. NAVAL HOSPITAL, Portsmouth, Va.—Drs. Donald Frank Fletcher, Jr., Horsey; Joseph Emory Mathias, Clarksburg, W. Va.; Garnett Bailey Moneymaker, Clifton Forge; Robert Albion Repass, Bristol.

U. S. NAVAL HOSPITAL, St. Albans, L.I., N.Y.—Dr. Virginius Archer Marks, Hopewell.

U. S. MARINE HOSPITAL, San Francisco—Dr. William Edward Newby, Richmond.

WALTER REED GENERAL HOSPITAL, Washington—Dr. Charles Lankford Randolph, Jr., Richmond.

BROOKE GENERAL HOSPITAL, San Antonio, Texas—Dr. Harold Thomas Turner, Norfolk.

Dr. Frances Nunley Milam Stoneburner, Sutherlin, no appointment named.

UNIVERSITY OF VIRGINIA, DEPARTMENT OF MEDICINE.

The final celebration of the University classes of 1951 started June 8th, with a meeting of the Medical Alumni and the graduation exercises for all departments were held on the 11th. Dr. Frederick Wilkie of New York, vice-president, presided at the alumni meeting in the absence of the president, Dr. Alfred R. Shands of Wilmington, Delaware, who was in Europe. Later Dr. Wilkie succeeded to the presidency and Dr. Carrington Williams of Richmond was made vice-president. Following the final exercises, President and Mrs. Darden were hosts to graduates, their relatives and friends at a reception at their home.

The graduates in medicine with hospital appointments are:

UNIVERSITY OF VIRGINIA HOSPITAL, Charlottesville—Drs. Charles Coleman Donegan, Jr., Newport News; Warren Dunlap Elliott, Scranton, Pa.; Clifford Henry Fix, Lynchburg; Austin Taber Hyde, Jr., Damascus; Herbert Claiborne Jones, Jr., Petersburg; James Walter Stone, Lynchburg; William Harold Stout, Charlottesville; Henry Morse Tanner, Jr., La Crosse; Richard Norvell Taylor, Charlottesville; William Puller Wharton, Lexington; Harry Robert Yates, Jr., Roanoke.

NORFOLK GENERAL HOSPITAL, Norfolk—Dr. Evan Hamilton Ashby, Jr., Remington.

DEPAUL HOSPITAL, Norfolk—Dr. Albert McCray Jones, Norfolk.

MEMORIAL AND CRIPPLED CHILDREN'S HOSPITAL, Roanoke—Dr. Russell Smith, Charlottesville.

KING'S COUNTY HOSPITAL, Brooklyn, N. Y.—Drs. Albert Harold Arenowitz, Newport News; Bernard Oseroff, Norfolk.

BROOKLYN JEWISH HOSPITAL, Brooklyn, N. Y.—Drs. Seymour Brenner, Brooklyn; Albert Isadore Strauss, Brooklyn.

NEW YORK HOSPITAL, New York—Dr. Frank Nash Bilisoly, III, Portsmouth.

POSTGRADUATE HOSPITAL, New York—Dr. Harvey Jerome Breit, Norfolk.

ST. VINCENT'S HOSPITAL, New York—Drs. Ira Marshall Cantin, Virginia Beach; Paul Sabak Derian, New York.

ST. LUKE'S HOSPITAL, New York—Dr. Thomas Mahlon Wright, Upper Montclair, N. J.

MORRISANIA CITY HOSPITAL, Bronx, N. Y.—Dr. Leonard Ned Ebin, New York.

STRONG MEMORIAL HOSPITAL, Rochester, N. Y.—Dr. Moffett Robson Walker, Jr., Petersburg.

UNIVERSITY HOSPITALS OF CLEVELAND, LAKESIDE HOSPITAL, Cleveland—Dr. Lewis Booker, New Castle, Del.

JEFFERSON-HILLMAN HOSPITAL, Birmingham, Ala.—Drs. John Chalkley Buchanan, Clintwood; Wilfred Qualls Cole, Jr., Jackson, Miss.; Charles Theodore Harris, Jr., Salisbury, N. C.; Allan Hammett Jefferies, Alexandria; Richard Bennett Meriwether, Sanford, Fla.; William Herman Pifer, Winchester; John Miles Stirewalt, New Market.

MEMORIAL HOSPITAL, Wilmington, Del.—Dr. Floyd Woodson Carneal, Jr., Hampton.

VIRGINIA MASON HOSPITAL, Seattle—Dr. Wallace Alexander Coburn, Seattle.

JAMES WALKER MEMORIAL HOSPITAL, Wilmington, N. C.—Dr. William Templeton Davis, Charlottesville.

BOWMAN-GRAY SCHOOL OF MEDICINE, Winston-Salem, N. C.—Dr. Frank Hutchinson Hamilton, Jr., Rock Hill, S. C.

EMORY UNIVERSITY HOSPITAL, Atlanta, Ga.—Dr. William Stribling Dingledine, Harrisonburg.

SINAI HOSPITAL, Baltimore—Dr. Adolphe Max Ehrenworth, Norfolk.

BALTIMORE CITY HOSPITALS, Baltimore—Dr. James Moultrie Lee, Savannah, Ga.

JOHNS HOPKINS HOSPITAL, Baltimore—Dr. Joseph Humphries Magee, Lexington.

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CHARITY HOSPITAL, New Orleans—Drs. John Breckinridge Gorman, Lynchburg; Edsel Ford Stewart, Osyka, Miss.

HOTEL DIEU, New Orleans—Dr. Jose A. Semidey, Charlottesville.

MASSACHUSETTS MEMORIAL HOSPITALS, Boston—Drs. James Dunn Mason, Jr., Petersburg; William Taylor Tucker, Franklin.

PETER BENT BRIGHAM HOSPITAL, Boston—Dr. Charles Henry Sackett, Lynchburg.

BOSTON CITY HOSPITAL, Boston—Dr. Foxhall Parker Thornton, Jr., Franklin.

PRESBYTERIAN HOSPITAL, Chicago—Dr. Robert Traylor Helmen, South Bend, Ind.

ST. LUKE'S HOSPITAL, Chicago — Drs. Durell Boyd Horsley, Schuyler; William Henry Sipe, Harrisonburg.

PHILADELPHIA GENERAL HOSPITAL, Philadelphia —Dr. Philip Kearney Hensel, Camp Hill, Pa.

GRADUATE HOSPITAL OF UNIVERSITY OF PA., Philadelphia—Dr. Philip George Spaeth, Philadelphia.

SEWICKLEY VALLEY HOSPITAL, Sewickley, Pa.—Drs. Lawrence Perry Hyde, Buchanan; Edward Jordan Stoll, Arlington.

BARNES HOSPITAL, St. Louis—Dr. William Francis McGuire, Clifton Forge.

BAPTIST MEMORIAL HOSPITAL, Memphis, Tenn. —Dr. Charles Samuel Miller, Elkton.

TAMPA MUNICIPAL HOSPITAL, Tampa, Fla.—Dr. Andrew Christmond Mitchell, Bristol.

ROPER HOSPITAL, Charleston, S. C.—Dr. Harry Hawthorne Robinson, Jr., Charlottesville.

GEORGE WASHINGTON UNIVERSITY HOSPITAL, Washington—Dr. Joseph Lee Sedwitz, Youngstown, Ohio.

UNIVERSITY OF OKLAHOMA, OKLAHOMA CITY HOSPITAL, Oklahoma City—Dr. James David Wilson, Clifton Forge.

GORGAS MEMORIAL HOSPITAL, Ancon, C. Z.—Dr. Anthony James Grieco, Charlottesville.

U.S. NAVY HOSPITAL, Pensacola, Fla.—Dr. Ratcliffe Cox Barnes, New Kent.

U.S. NAVAL HOSPITAL, Portsmouth—Drs. Mayland Richard Bennett, Charlottesville; Raymond Keith Brown, Warrenton.

SAN FRANCISCO MARINE HOSPITAL, San Francisco—Dr. Cyrus Creston Farrow, Jr., Richmond.

STAPLETON MARINE HOSPITAL, Staten Island, N. Y.—Drs. Francis Gordon Griffin, Windsor; Robert Harris, Charlottesville.

U.S. NAVY HOSPITAL, San Diego, Calif.—Dr. Robert Breece Pennington, Nathan's Creek, N. C.

U.S. MARINE HOSPITAL, New Orleans—Dr. Roy Preston Sandidge, Jr., Charlottesville.

WILLIAM BEAUMONT ARMY HOSPITAL, El Paso, Texas—Dr. Lewis Willard Shankel, Charlottesville.

Alumni Association, Medical College of Virginia.

Mr. E. Claiborne Robins, Richmond, an alumnus of the School of Pharmacy, succeeded Dr. Donald S. Daniel of Richmond, as president of the Alumni Association. Dr. H. Hudnall Ware of Richmond was named president-elect, and the following were named vice-presidents: Dr. Brock D. Jones, Norfolk, Dr. W. A. Ratcliffe, Richmond, W. Roy Smith, Petersburg, and Miss Julia Watkins, Richmond. Mr. J. Curtis Nottingham of Richmond was renamed secretary and Dr. Harvey B. Haag, also of Richmond, treasurer.

Dr. Coleman Resigns.

Dr. Claude C. Coleman, a pioneer in neurosurgery in the United States, resigned his position with the Medical College of Virginia, effective July 1. He was professor and head of the department of neurological surgery, where he is completing forty-two years of service with the College. During this time he has been honored by many other organizations.

Dr. Alfred R. Shands, Jr.,

Has been appointed as a member of the Armed Forces Medical Policy Council effective July 1, 1951. Dr. Shands is Medical Director of the Nemours Foundation and Surgeon in Chief of the Alfred I. duPont Institute of Wilmington, Delaware. He has been a civilian medical consultant to the Surgeon Generals of the three services.

A graduate of the University of Virginia's Medical School, Dr. Shands is a former Professor of Surgery at Duke University. He is a visiting Professor in Orthopedic Surgery at the University of Pennsylvania. During World War II, Dr. Shands served as a colonel in the U. S. Army Medical Corps, and continues to hold that rank in the Organized Reserve Corps.

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Dr. Maxwell E. Lapham,

Dean of the Tulane Medical School, New Orleans, has gone to South America to survey medical facilities there. The survey is being sponsored by the United States State Department and he will represent the Association of American Medical Colleges and Universities. Dr. Lapham was at one time field clinician in obstetrics of the Department of Clinical and Medical Education of the Medical Society of Virginia.

A.M.A. Cancels Session in Houston.

The Winter Clinical session of the American Medical Association will not be held in Houston, as originally scheduled, but has been switched to Los Angeles. This is due to the fact that the huge San Houston Coliseum where the exhibits were to be held is tied up by a 10-year contract and cannot be secured between October 1 and April 1.

The Gill Memorial Eye, Ear and Throat Hospital,

Roanoke, has just completed its twenty-fourth Annual Spring Congress in Ophthalmology and Otolarygology. The total registration was 350. Registrants came from 38 states and all parts of Canada. Twenty-three guest lecturers, comprised the faculty. The twenty-fifth Congress will be held in Roanoke, April 1-8, 1952.

News from Department of Medicine, University of Virginia.

Dr. S. W. Britton, Professor of Physiology at the University, has been appointed Visiting Professor of Endocrinology at the University of Nigeria, British West Africa, and will leave for Africa in February, 1952. Announcement of the award of the visiting professorship was made by the Department of State under terms of the Fulbright Act. During his stay in Africa, Dr. Britton will continue his investigations on endocrine involvement in maintenance of the upright position, using primates available in the region. Dr. Britton will be on leave of absence from the University during the second semester and will retire at the end of the year, after 24 years of service.

Dr. George Cooper, Jr., Associate professor of Roentgenology, was awarded the American Cancer Society's 1951 Medal, May 18, in recognition of his contributions to the control of cancer. Dr. Cooper is medical and scientific director and a member of the Board of Trustees of the Virginia Division, American Cancer Society, and chairman of the cancer committee of the Medical Society of Virginia. Last year's American Cancer Award for Virginia was presented to Dr. Edwin P. Lehman, Professor of Surgery and Gynecology, University of Virginia.

The new Virginia Hearing Foundation at the University is being organized under the direction of Dr. Fletcher D. Woodward, Professor of Otolaryngology, and Dr. James M. Mullendore, director of the Speech and Hearing Center at the University.

The Virginia Hearing Foundation will offer a training center for specialists in the diseases of the ear and will provide a complete diagnostic clinic. Future plans include the setting-up a full-time traveling clinic, which will travel throughout Virginia conducting hearing tests.

Dr. Fletcher D. Woodward, Professor of Otolaryngology at the University, retires as Chairman of the School of Otolarygology July 1, after 26 years of service, but will remain active on the teaching and clinical programs. Dr. G. Slaughter Fitz-Hugh, clinical assistant professor of otolaryngology, will succeed Dr. Woodward as chairman of the Department.

Dr. Halsted S. Hedges, Emeritus Professor of Ophthalmology at the University of Virginia, was honored for his 52 years of service by the Virginia Society of Ophthalmology and Otolaryngology at its 32nd annual meeting May 12 in Roanoke. Dr. Hedges, the first member of the Society to be so honored, has been a member of the medical faculty of the University of Virginia since 1899.

Dr. Douglas E. Bragdon, assistant professor of Anatomy at the University, has received a grant from the Committee on Research in Problems of Sex, of the National Research Council, for studies on the corpora lutea of ovoviviparous snakes.

The Tidewater Ophthalmological and Otolaryngological Society

Held its second meeting in Norfolk, May 23rd. The guest speakers were: Dr. P. N. Pastore, Professor of Otolaryngology at the Medical College of Virginia, who spoke on the effects of quinine poisoning of the inner ear of rats and rabbits, based on experiments. Dr. W. W. Zimmerman of the staff

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of the Medical College of Virginia read a paper entitled "Causes of Blindness".

Dr. C. C. Cooley is President and Dr. John G. Sellers Secretary-Treasurer of this Society. Both are of Norfolk.

News from State Health Department.

Dr. J. B. Porterfield has resigned his post as an assistant director of local health services with the State Health Department, to become a senior medical officer with the Veterans' Administration in Johnson City, Tennessee.

Dr. C. P. Pope has returned from educational leave and will again assume his duties as Health Officer of the Pulaski-Wythe Health District, with headquarters in Pulaski.

Dr. W. R. Payne

Of Newport News announces the association of Dr. W. James Baggs, Jr. in the practice of gynecology. Dr. Baggs is a graduate of the Medical College of Virginia. He served his internship and residency in obstetrics and gynecology on the Tulane Service of Charity Hospital, New Orleans. He was a Lt. j.g. in the U. S. Navy Medical Corps during World War II, participating in several invasions with the Marine Corps in the Pacific.

The American College of Surgeons

Will hold its 37th annual Clinical Congress in San Francisco, November 5 to 9, 1951, with headquarters at the Fairmount Hotel and Civic Auditorium. The thirtieth annual Hospital Standardization Conference is scheduled to be held concurrently, with meetings in the Civic Auditorium, as a part of the Congress. The combined programs will include scientific and technical exhibits, color television, cine clinics, medical motion pictures, scientific sessions, panel discussions, conferences, symposia, official meetings, and forums. Several thousand surgeons and hospital representatives are expected to attend.

Dr. Arthur W. Allen of Boston is Chairman of the Board of Regents of the College; Dr. Henry W. Cave of New York is President, Dr. Alton Ochsner of New Orleans is President-elect and will be installed as President on November 5; Dr. Paul R. Hawley of Chicago is the Director. Dr. Emile Holman is Chairman of the San Francisco Committee on Arrangement.s The hospital conference program is in charge of Dr. Paul S. Ferguson, Acting Assistant Director in charge of Hospital Standardization.

Dr. Ralph S. Faris,

Richmond, succeeded to the presidency of the American Institute of Homeopathy at its annual meeting at Atlantic City, June 17-21.

Married.

Dr. Robert Finley Gayle, III, and Miss Jane Lowry Dale, both of Richmond, June 16.

Dr. John L. Jennings,

Recently of Danville, has moved to Palmetto, Florida, where he will be engaged in the practice of medicine and surgery.

The Virginia Society for Pathology and Laboratory Medicine

Held its Spring meeting in Richmond on May 19, under the presidency of Dr. W. D. Nolan of Arlington.

1. *Scientific Program:* Dr. James E. Ash, Col. U.S.A., Ret., conducted a slide seminar on lesions of the ear, nose and throat.

2. The business meeting included the following committee reports:

a. *Committee on Laboratory Fees:* Dr. Palmer, chairman, had prepared a survey of laboratory fee schedules in the state and copies of his report were distributed to the members. A similar survey on salary rates for laboratory personnel will be made by the same committee, the survey to include the salary schedules of state and federal institutions.

b. *Committee on Resident Training:* Dr. Strauss, chairman, reported that the committee held a meeting in Charlottesville and submitted the unanimous report of the committee:

"The committee feels (1) that a minimum of three years supervised training is necessary in either pathological anatomy or clinical pathology as separate qualifications.

(2) that as combined qualifications three years in pathological anatomy and two years in clinical pathology may be adequate.

(3) two years training in pathological anatomy is insufficient and inadequate."

There was a lively discussion. The general feeling was that lengthening the training period might reduce even further the number of doctors interested

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in pathology and that one year in clinical pathology was certainly insufficient. On the other hand, it was generally agreed that two years in pathologic anatomy were not long enough either. Dr. Williams made a motion which was unanimously carried that the report of the committee be accepted and copies of the report be sent to the College of American Pathologists and to the American Board of Pathology.

c. *Committee on Evaluation of Laboratory Procedures:* Dr. Dolan, chairman, reported on his negotiations with Dr. Sunderman, who expressed his willingness to supply standards to all hospitals who were willing to participate and to distribute anonymous lists with the results obtained. It was felt that this matter should be handled through an outside agency. Dr. Williams made a motion that the committee complete the negotiations with Dr. Sunderman and put the evaluation in operation pending the expected national evaluation program of the American College of Pathologists. The motion was carried by unanimous vote.

d. *Committee on Tumor Registry:* Dr. Mann, chairman, reported that participation had been very poor. He offered to have blocks cut at his institute for distribution of slides among the Tumor Board and others interested in the material. It was felt that not only diagnostic problems but also cases of general interest should be sent to the committee.

e. *Committee on Local Arrangements For Regional Meeting:* Dr. Scherer, chairman, asked for scientific papers for members of the Society for the morning session. Titles should be submitted as soon as possible. Selection will be made by the program committee consisting of Drs. Mann, Scherer, Rawson and Bobbitt.

Applications for Membership: Application for membership by Dr. K. F. Menk of Falls Church, Virginia, was presented and accepted unanimously.

M. L. DREYFUSS, M.D.
Secretary-Treasurer.

The United States Chapter of the International College of Surgeons

Will hold its 16th annual assembly in Chicago on September 10th through the 13th, 1951, with headquarters at the Palmer House. An excellent program has been arranged. Prominent surgeons from the United States and other countries will participate. Scientific sessions will be held by all specialty sections of the United States Chapter.

The annual banquet will take place on Wednesday evening, September 12. Mr. Lawrence Abel, F.R.C.S. (Eng.) of London, will be the principal speaker. The Assembly will conclude with the convocation, to be held in the Civic Opera House on the evening of September 13. Senator Estes Kefauver will deliver an address on "The America of Tomorrow."

Hotel reservations may be arranged by writing to the Housing Division, Chicago Convention Bureau, 33 North LaSalle Street, Chicago 2, Illinois.

Dr. Hal S. Johnson

Announces his removal to 22 Church Street, Martinsville, where he is engaged in private practice limited to the eye, ear, nose and throat.

Dr. E. G. Gill

Roanoke, read a paper by invitation on "The Surgical Treatment of Glaucoma" at the Second Clinical Conference of the Alumni of the New York Eye and Ear Infirmary, New York City, May 10th and 11th. Dr. Gill was also elected president-elect of the Alumni Association and a member of the Executive Council.

Office Space For Rent.

Doctor's or Dentist's office available immediately, corner of Harrison and Franklin Streets, Richmond, Call 5-4387. (*Adv.*)

OBITUARIES

Dr. Lockburn B. Scott,

Former secretary and treasurer of the Norfolk County Medical Society for twenty-seven years, died at the home of a daughter in Norfolk, May 17th, at the age of 89. He was a native of Ontario, Canada, and received his medical degree at the Manitoba Medical College in 1907. He retired from active practice about fifteen years ago. He is survived by three daughters.

Dr. Eugene Knight Bowles,

Retired physician of Jordan Mines, died May 18, at the age of 68 years. He was a graduate of the Baltimore Medical College in 1907 and had practiced in Alleghany County for thirty-five years. He was a former member of the county's welfare and electoral boards and health department. His wife and several children survive him.

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